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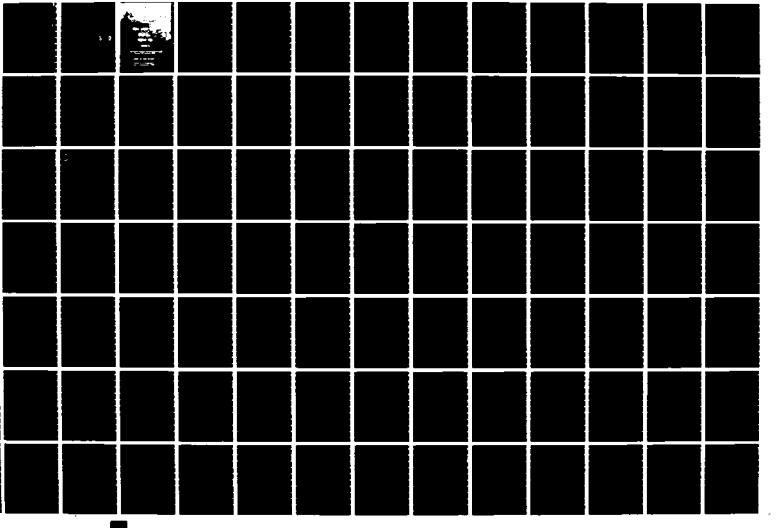

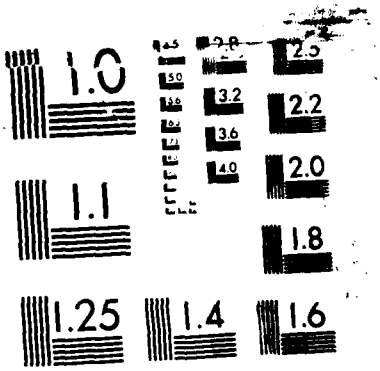

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Results of Year 2 are presented and includes: (a) the theoretical rationale and strategy for analysis of neuromotor coordination mechanisms in fast limb movements; (b) a progress report of experimental and modelling studies conducted during Year 2; (c) an article describing an EMG-level mathematical model of fast arm movement; (d) an article describing the stability of delay equations for simple human stretch reflexes; (e) an article describing

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theoretical bases for the sensory imparted learning model; (f) an article describing the prediction of male and female isometric arm strength through anthropometric measures; (g) experimental results of gender differences and muscle fatigue effects upon speed of neuromotor coordination mechanisms; and (h) experimental results of muscle fatigue upon speed of movement and the effects of tonic vibratory response upon neuromotor coordination mechanisms.

Coordination Mechanism In Fast Human Movements - Experimental And Modelling Studies

VOLUME I

ANNUAL SUMMARY REPORT

Walter Kroll
William L. Kilmer

FEBRUARY 1982

SUPPORTED BY

U. S. Army Medical Research and Development Command
Fort Detrick, Maryland 21701

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TABLE OF CONTENTS

Introduction

The present study is investigating the basic neuromotor coordination mechanisms involved in a rapid elbow flexion movement, and in developing mathematical models to explain the interaction of these basic neuromotor coordinations with the biomechanical parameters of movement speed. Speed of movement is being assessed in a biomechanics mode via displacement, velocity, acceleration, point of inflection between acceleration and deceleration, and the total time of an elbow flexion movement. Electromyographic analysis techniques are used to monitor the sequential timing of agonist and antagonist muscle activity. The mathematical modelling effort incorporates the biomechanical parameters into an interface with the neurophysiological parameters involving the central and peripheral nervous systems, and then extends the interface to include viscicelastic perperties of the muscle, activation delays, and neuronal pools.

The experimental approach includes consideration of the neuromotor coordination mechanisms in both loaded and unloaded elbow flexion movements; changes in control mechanisms due to practice and learning effects; changes in control mechanisms due to local muscular fatigue induced by isometric exercise in the agonist and in the antagonist muscle groups; feasible training and practice regimens involving artificial means of enhancing beneficial changes in control mechanisms; and development of suitable mathematical models to explain in functional terms the ways in which the adaptive mechanisms can account for changes in basic coordination skill and the breakdown of skilled movement patterns due to local muscular fatigue. The planned series of studies incorporates research protocols from at least three usually distinct and isolated areas of research: neurophysiology, biomechanics, and computer science and mathematical modelling.

Summary of Part I. Experimental Studies

The first formal investigation completed December, 1980 was designed to secure criterion measure estimates essential for input into preliminary mathematical modelling attempts for an unloaded fast elbow flexion movement. As the early mathematical models developed, it was necessary to revise experimental methodologies and to collect additional criterion measures as dictated by modelling needs. The first stage of mathematical modelling has been completed and is described in Part II of this report.

Beginning in January, 1981 the second formal investigation was begun. Again guided by the insights and requirements generated by the mathematical modelling, the second study assessed basic neuromotor mechanisms involved in fast elbow flexion: (a) in loaded and unloaded conditions, (b) under conditions of rested and induced fatigue of the agonist or the antagonist muscle groups, (c) a unique condition in which agonist and antagonist muscle groups are, simultaneously fatigued, and (d) in male and female subjects.

During the second year of the grant, data were collected on make and female subjects trained on isotonic rapid elbow flexion movements. Following sufficient training to yield reproducible motion characteristics, muscle fatigue was induced separately for agonist and antagonist muscles as well as for a combined agonist and antagonist muscle fatigue regimen. Both high and low intensity loads were employed to produce isotonic fatigue. Testing was done in the loaded and unloaded modes. Biomechanical data on displacement, velocity, acceleration, and point of inflection between acceleration and deceleration was obtained. Concurrent EMG activity of agonist and antagonist muscle groups was monitored. The results are being used to further refine mathematical models of fast arm

movement as driven by volitional commands that were derived during the first year of the grant. Pilot studies were initiated to evaluate the feasibility of using functional electrical stimulation to increase speed of limb movement, aid in identifying mechanisms of neuromotor coordination, and examine the likelihood of imparting movement patterns artificially without actual physical practice.

Part I-A. Completed Work

Both Principals Investigators devoted full time during the first summer to the grant by (a) identifying, reviewing, and evaluating recent research publications relevant to theoretical aspects of the grant project, (b) planning the revised research protocols in detailed methodology, and (c) identifying, reviewing, and evaluating recent research publications relevant to data analysis and computer programming. Two research assistants were employed during the month of August to expedite these undertakings. Preliminary orders for necessary expendable supplies were sent out.

In September, 1980 additional graduate research assistants (RA's) were recruited. In Dr. Kilmer's case the RA continued on with computer programming for the mathematical modelling aspects of the project. In Dr. Kroll's case new RA's were indoctrinated into research data collection protocols. The indoctrination phase included practice in apparatus usage, maintenance, and calibration techniques as well as pilot testing research measurement schedules to insure reliability of data collection. The indoctrination phase lasted six weeks resulting in highly proficient research assistants capable of collecting reliable data.

Beginning the end of October, 1981 data collection for the first project began. The first study was designed to secure essential criterion measure necessary for input into preliminary mathematical models. These criterion measures include:

...maximum isometric strength of elbow flexion and extension

...rate of tension development

...integrated electromyographic activity at maximal and sub-maximal tensions and joint angles

...agonist-antagonist contraction patterns during unloaded speed-of-movement elbow flexion

...effects of local muscular fatigue upon neuromotor coordination

Preliminary mathematical modelling attempts quickly showed that additional criterion measures would be necessary. As a result of the modelling requirements, upper limb volumes and biceps and triceps reflex time and rate of muscle tension development were collected. Limb volumes were estimated by anthropometric and water displacement techniques. The research assistant was trained in these techniques by Professor Frank Katch of the Department of Exercise Science who is a recognized researcher in the body composition area. A MEDLARS literature search revealed only a few scattered articles dealing with upper limb reflex times with none of the data appropriate for modelling requirements. Suitable apparatus was designed and built for upper human limb reflex testing. Limb volumes and reflex times have been collected on all subjects tested to date.

In this first study of Phase I, each of the 12 subjects was tested over ten experimental sessions with each session taking 90-120 minutes. Eight of these sessions involved speed of movement testing while the other two sessions involved upper limb reflex and volume assessments. Each of the first four experimental days included 50 trials of rapid elbow flexion speed of movement

to a designated target. Previous research has shown that this amount of practice insures that all subjects become well-practiced and exhibit consistent, stabilized performance both in the speed of movement as well as in the neuro-motor coordination mechanisms. Following the establishment of well-practiced performance, local muscular fatigue was induced by two different effort/rest exercise regimens. A 5-second maximal isometric contraction with a 5-second intertrial rest period--designated the 5:5 condition--for a total of 30 serial trials constituted the more intense fatigue series. A 5-second maximal isometric contraction with a 10-second intertrial rest period--designated the 5:10 condition--for a total of 30 serial trials constituted the less intense exercise regimen.

These two exercise regimens--the 5:5 and the 5:10 conditions--were designed to produce different levels of fatigue in the involved muscle group. The 5:5 and the 5:10 exercise regimens were administered on separate occasions to the agonist (elbow flexors) and to the antagonist (elbow extensors) muscle groups using a balanced order order of presentation over subjects and across test sessions to minimize contaminating test order effects.

One of the purposes of inducing high and low intensity local muscular fatigue in the agonist or antagonist muscle groups was to ascertain the role of changes in the peripheral muscle state and in peripheral muscle afferent feedback to higher nervous system centers. Specifically, the question of whether or not programmed central commands for a fast ballistic movement can be altered by the presence of different levels of muscular fatigue.

I-B. Work in Progress

In Phase II of the grant proposal an investigation using the same testing schedule outlined for the Phase I study has been replicated for elbow flexion

speed of movement. However, to further elucidate the basic neuromotor coordination mechanisms a load is imposed upon the limb movement task. The imposition of a load, of course, greatly affects the muscle activation time and sequential firing of involved muscle groups and constitutes a more complex movement task with distinctly more complex nervous system control system involvement.

In order to insure identical loading for subjects with different limb lengths, an inertial loading technique was used. The natural moment of inertia can be altered by placement of light weights at long distances and heavy weights at short distances from the fulcrum point. The protocol employed utilized a fixed weight for all subjects but varied the distance from the fulcrum point to comply with the condition of imposed moment of inertia. One load condition used was equal to two times the natural moment of inertia while the second load condition was equal to five times the natural moment of inertia.

In addition to the two different exercise regimens of 5:5 and 5:10 administered to the agonist and to the antagonist muscle groups on separate occasions, another set of exercise regimens was also administered. Based upon the mathematical modelling considerations, it was of interest to ascertain the effect of local muscular fatigue in <u>both</u> the agonist <u>and</u> antagonist muscle groups simultaneously upon basic neuromotor coordination mechanisms. To accomplish such a goal of producing fatigue in both agonist and antagonist muscle groups, a flexion-to-extension contraction sequence was designed.

By using a 5-second contraction of the elbow flexors followed immediately by a 5-second contraction of the elbow extensors with no intertrial rest period, fatigue effects equivalent to 5-second contraction and 5-second rest periods

were produced in both the agonist and antagonist muscle groups. Similarly, if the effort sequence is a 5-second contraction of the elbow flexors followed by a 5-second contraction of the elbow extensors followed by a 5-second rest period, the equivalent fatigue effects of a 5-second contraction, 10-second rest period exercise regimen can be produced in both the agonist and antagonist muscle groups simultaneously.

These two new exercise regimens, designated the 5/5:0 and the 5/5:5, aloow assessment of fatigue effects in the agonist and the antagonist muscle groups upon basic neuromotor coordination mechanisms and provide a stringent test of the predictive power of the mathematical model being developed. In actuality the two new exercise regimens produced slightly more fatigue than the 5:5 and the 5:10 regimens upon which they were based because some degree of co-contraction of agonist and antagonist muscle groups occurs in maximal isometric contractions. Such co-contraction effects, however, are desirable since they will heighten the local muscular fatigue produced.

The Phase II study described above involved 12 male and 12 female subjects with each subject attending 12 test sessions of 90-120 minutes each. Ten of these sessions involved speed of movement and exercise conditions while the other two sessions were for upper limb reflex and volume testing. One of the research assistants (Zulma C. Garcia) used some of the data for her Ph.D. dissertation in exercise science under the chairmanship of PI Kroll. Data collection was completed March, 1981 with subsequent reduction and analysis of data taking place. A Master's thesis was completed as a pilot study dealing with the effects of vibration upon speed of movement and neuromotor coordination mechanisms. This graduate student, Marilyn Teves, was not a research assistant but her work was supported in part by purchase of essential expendable supplies.

Data collected during Year 1 (1 July 1980-1 June 1981) was reduced, analyzed, and results inputted into mathematical modelling projects. Research manuscripts suitable for publication in scientific journals have been prepared (see Appendices). Based upon insights generated by the mathematical modelling, refinements in studies planned for Year 2 became necessary as they were also during Year 1. Independent of the analysis of data all ready collected and being inputted into mathematical modelling projects, Year 2 experimental studies were begun the summer of 1981.

Essentially the same testing schedule which replicated fatigue and coordination studies during Year 1 was begun the summer of 1981. The exercise regimens, however, utilized isotonic rather than isometric muscular contractions to induce fatigue.

Testing Schedule

Unloaded Movement Coordination and Fatigue

Subjects will perform 50 trials daily of rapid elbow flexion speed of movement. Four days of practice will be necessary to produce consistent and stabilized performance. Following establishment of stabilized performance of the neuromotor control mechanisms and motion characteristics, local muscular fatigue will be induced as per the following exercise regimens:

1. Agonist Muscle Fatigue

   Six bouts of isotonic elbow flexion. During each bout subjects will perform repetitions at a load equal to that necessary to produce a six repetition maximum (6-RM) or a 20-repetition maximum (20-RM) bout. Preliminary testing on subject during the baseline stabilization days will be used to establish the individual loads to be used to produce a high intensity (6-RM) and low intensity (20-RM) fatiguing exercising regimens.

2. Antagonist Muscle Fatigue

Same protocol as 1 above.

One of the purposes of high and low intensity local muscular fatigue via the isotonic exercise regimens on the agonist and antagonist muscle groups separately is to ascertain the role of changes in peripheral muscle feedback to higher nervous system centers and determine if programmed central commands for a fast ballistic movement can be altered by fatigue. Isotonic and isometric exercise regimens produce different kinds of fatigue and comparisons of the isotonic and isometric exercise regimen effects upon neuromotor coordination mechanisms is of particular relevance to understanding coordination of a fast movement.

Loaded Movement Coordination and Fatigue

The same testing schedule proposed above will be replicated for elbow flexion speed of movement against a load. In order to insure identical loading for subjects with different limb lengths, an inertial loading technique will be used. The protocol will utilize a fixed weight for all subjects and will vary the distance from fulcrum point to comply with the condition of moment of inertia. One load condition will equal two times the nature moment of inertia and the second load condition will equal five times the natural moment of inertia.

Functional Electrical Stimulation

Pilot studies were initiated to evaluate the feasibility of using electrical stimulation to increase speed of fast limb movement and examine the possibility of imparting movement patterns artificially without actual physical practice. Study of the literature on Funcational Electrical Stimulation (FES)

coupled with results from our own pilot studies showed that no electrical stimulation apparatus was commercially available which possessed all of the control features needed. Extended negotiations with several manufacturers eventually led to one manufacturer agreeing to make necessary modifications to their available stimulator. Design problems still exist with the present simulator and additional modifications are being made by our own electronic technician.

Reverse Look Theory of Motor Learning

As a direct result of Year 2 efforts, a new theory of motor learning has been formulated anf formally presented at a neurophysiology symposium on movement disorders held at Columbia University in March of 1982 (see Appendix for paper). The new theory has been named the reverse loop theory of motor learning since it is based upon sensory imparted learning (SIL) produced by electrical stimulation of movement synergy patterns prescribed by an analysis of neuromotor coordination mechanisms of human limb movement. One article is being prepared describing the reverse look theory of motor learning and sensory imparted learning for publication in a suitable journal, possible the Journal of Motor Behavior of Experimental Brain Research.

II. Proposed Experimental Studies - Year 3

At the present time we are listing the effectiveness of several aspects of electrical stimulation.

1. Wave form: monophasic, biphasic, biphasic compensated
2. Pulse duration
3. Frequency of pulses
4. Intensity level

The problem is made more complex because of the likelihood that motor units of different muscle fiber type composition may require different stimulus train parameters. Current efforts in mathematical modelling are directed at this problem and will provide important guidelines for stimulation train parameters. Even at this time, however, crude parameter values have been ascertained for effective stimulation and continued work will serve to refine the techniques and no obstacle to further progress is anticipated.

Early pilot testing revealed that electrical stimulation to produce muscle synergy contraction as prescribed by a model of neuromotor coordination mechanisms can produce learning (see appendix for paper presented at Columbia University Neurophysiology Symposium on movement disorders). Studies proposed for Year 3 will examine the importance of neuromotor coordination mechanisms as far as producing improved performance through the use of electrical stimulation.

Our work to date has shown several important neuromotor coordination mechanisms are involved in fast limb movement:

1. agonist to antagonist latency of activation
2. duration of EMG burst (or envelope of EMG activity) in agonist and antagonist modes
3. relative EMG intensity of agonist and antagonist muscle activity

Series 1. A series of activities will manipulate all three basic neuromotor coordination mechanisms simultaneously and assess the degree to which well-practiced speed of limb movement can be improved by electrical stimulation designed to simulate muscle synergy contraction patterns. By optimizing the three neuromotor coordination mechanisms to produce faster limb movement the effectiveness and magnitude of electrical stimulation regimen can be assessed.

Series 2. In order to elucidate the relative importance of each of these three major neuromotor coordination mechanisms to limb movement, electrical

stimulation regimens must be devised to produce sensory imparted learning which manipulates each of these brain control mechanisms. Following the establishment of well-practiced, stabilized speed-of-movement times and associated EMG patterns, groups of subjects will be electrically stimulated while optimizing only one of the three basic control factors as a means of improving performance. One group of subjects, for example, will be stimulated electrically with only the agonist to antagonist latency of activation manipulated to optimize limb movement. Another group of subjects will be stimulated with the duration of EMG activity manipulated to optimize limb movement. A third group of subjects will be stimulated with the relative EMG activity of agonist and antagonist muscles being manipulated to optimize limb movement. These three groups will be compared to a control group.

Series 3. A third series of studies will assess the relative importance of the three basic neuromotor coordination mechanisms taken two at a time; i.e., electrical stimulation will be applied to produce a muscle synergy contraction pattern which optimizes two of the three basic control mechanisms. Such a paradigm requires three treatment conditions: 1 and 2, 1 and 3, 2 and 3 with comparison against a control group.

Each of these three series of electrical stimulation studies will be guided by actual experimental results and by results of the optimization studies of mathematical modelling. The exact research methodology must remain flexible within these constraints, especially for series two and three. It is impossible to anticipate when all three series of studies can be completed, but it is unlikely that they can be completed in one year and a request for an additional year of grant support is a distinct likelihood. Stabilization of limb movement

time and EMG patterns requires extensive practice for one or more weeks. Electrical stimulation treatment sessions must be given for at least 2-3 weeks before a re-test, and several such 2-3 week sessions must be given consecutively in order to determine the magnitude of effects.

We would also like to begin to investigate the potential of high frequency electrostimulation purported by the Russians to produce better strength gains than normal exercise regimen. Our intention is to utilize the electromuscular stimulator (EMS) now commercially available to determine if the neuromotor coordination mechanisms as expressed in EMG patterns are affected by increases in muscle strength produced by EMS. Such a pilot investigation can be accomplished most effectively in connection with the proposed coordination studies. Our equipment request reflects the cost of ~~ten~~ two such Electrostim 180 models manufactured by the NU-Med Surgical Supply Co. ($3,250 each).

PART II

Modelling

Summary of Part II:

We describe a two-compartment model of the neuromuscular system involved in the voluntary fast arm movement to a target discussed by Kroll in Part I. The first compartment accepts averaged biceps and triceps EMG signals as inputs, and models the arm's musculo-skeletal response by producing elbow angular position $\phi(t)$ and velocity $\dot{\phi}(t)$ over the corresponding movement time. This is called our E/Q/$\dot{\phi}$(t) model, and its defining equation is of the form

$$\dot{\phi}(t) = (\text{extensor torque} = Q_E) - (\text{flexor torque} = Q_F).$$

A detailed version of this equation is being computer-simulated with the PASCAL programme listed in Appendix A.

Our second compartment, called our com/cont/E model, accepts volitional commands as inputs, and models the nervous system's response by producing smoothed flexor and extensor EMG signals to feed into the E/Q/$\dot{\phi}$ model. Fig. II-4 gives a schematic representation of the com/cont/E model, which is explained in detail in the report.

The purpose of our two models is to separate the various control functions in the ballistic arm-movement system well enough to understand where adaptation occurs as speed, precision, and coordination of movement improve with practice.

This is the first publication that has arisen from work on this contract.

II-A Completed Work

Since last June we have completed an initial specification of two models that will help us to better understand and guide the direction of Kroll's experiments. The first model accepts smoothed EMG inputs to flexor and extensor muscles and calculates arm angle dynamics for the fast arm movement described above by Kroll. This model is denoted the E/Q/$\dot{\theta}$(t) model (short for EMG/elbow torque = Q/elbow angular velocity dQ/dt = $\dot{\theta}$). The second model feeds flexor and extensor smoothed EMG outputs into the first model, and receives its input commands from a volitional fast arm movement center in the brain, presumably via the pyramidal tract from the cerebellum to the brainstem and spinal cord (cf. Miles and Evarts, 1979). This model is denoted the Com/Cont/E model (short for command/neural control/EMG). The appropriate connection of our two models provides a complete overall model for the fast arm movement as driven by volitional commands.

Below we describe the E/Q/$\dot{\theta}$ model first and the com/cont/E model second. Section II-B then proposes further studies for next year.

1) The E/Q/$\dot{\phi}$ model

This model arose out of a desire to formulate a quantitative account of the E/Q/$\dot{\phi}$ part of the arm movement to target described by Kroll. After reviewing the relevant literature, we rejected possible model formulations at the molecular biophysical level as too complex (cf. Hatze, 1978, 1980, and Dijkatra et al., 1973a, 1973b). In addition, for our movement, they would be too difficult to specify quantitatively. We also rejected model formulations at the spring-mass postural level (cf. Sakitt, 1980) because they would be too steady-state oriented to allow an analysis of our large transient arm motion. Other control engineering models (cf., e.g., McRuer) seemed too general or too coarse-grained for our purpose.

Figure II-1 gives a schematic representation of the framework for the E/Q/$\dot{\phi}$ model we chose. Its corresponding top-level analysis formula is

$$J\ddot{\phi} = Q_E - Q_F \ , \qquad (1)$$

where each dot over ϕ indicates a time derivative, and Q_E and Q_F are the respective torques about the elbow as exerted by the extensor and flexor groups of muscles (all the muscles in each group operate in approximate temporal and tensional unison throughout our movement (Lagasse, 1975)). J is the arm's moment of inertia for rotations about the fixed elbow joint as shown in Fig. II-1 (over a ϕ change from 60° to about -30°).

At the second level of analytical refinement, we let

$$Q_E = \psi_{VE} \cdot \psi_{IE} + \tau_E \qquad (2)$$

where τ_E is the torque due to viscoelastic braking of the movement (Lestienne, 1979), and ψ_{IE} is the maximum (over $-90° \leq Q \leq 90°$) isometric torque producable by the extensor, given the smoothed extensor EMG envelope,

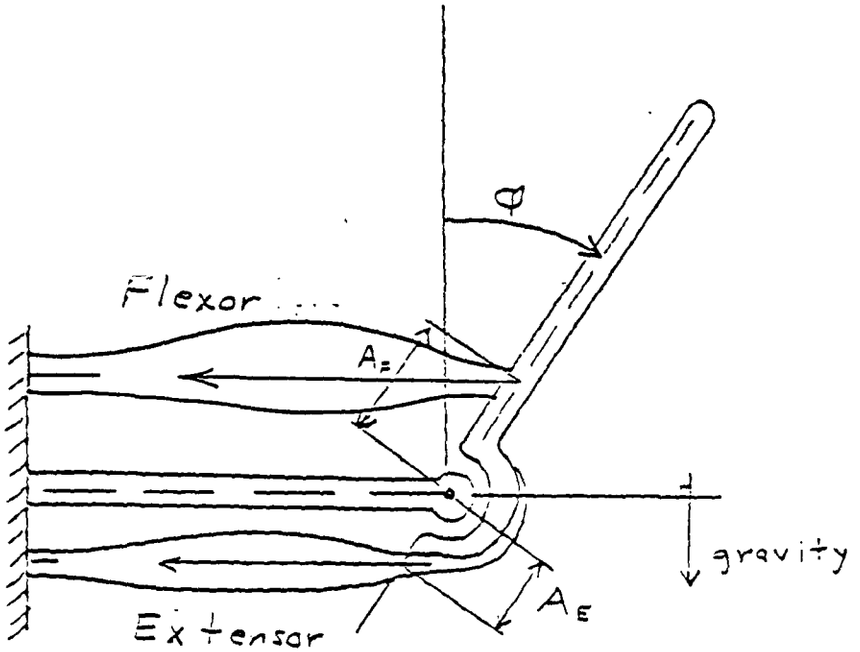


Figure II-1. Schematic for the framework of $\dot{E}/\dot{Q}$ model. See text for explanation.

$\overline{EMG}_E$, recorded over the last second (250 ms, or several seconds would serve as well, but we need a standard). To find ψ_{IE}, we accept the claim of Messier, et al., 1971, that under static loads, "the averaged EMG is directly proportional to muscle tension and the constant of proportionality is independent of muscle length." Admittedly, the force properties of muscles undergoing rapid active or passive stretch or compression are very different than when these muscles are being actively or passively subjected to static loads. But from what little is known, the best we can do to account for this is to include the ψ_{VE} factor appearing in (2). ψ_{VE} is a fraction between 0 and 1 that inserts the force-velocity relationship suggested by Thorstensson et al., 1976, and Perrine and Edgerton, 1978, for the human knee, and by Lagasse's 1975 observations on forearm accelerations during maximally fast movements (quite like our experimental ones only untargeted).

Our ψ_{VE} function is

$$\psi_{VE}(\dot{\phi}) = \left[\!\left[1 - \frac{\dot{\phi}}{1000} \right]\!\right] \tag{3}$$

where $\dot{\phi}$ is in degrees/sec, and the double-cusped brackets mean

$$\left[\!\left[x \right]\!\right] = \begin{cases} x \text{ if } x \geq 0 \\ 0 \text{ otherwise} \end{cases}$$

For the flexor side we let

$$Q_F = \psi_{VF} \cdot \psi_{IF} \tag{4}$$

where ψ_{IF} is similar to ψ_{IE} and $\psi_{VF} = \psi_{VE}$. Equating ψ_{VE} to ψ_{VF} for our movement is inaccurate to some degree because the flexor undergoes active contraction and/or passive compression whereas the extensor undergoes passive or active stretch (actually, the joint must be stabilized so there are no purely passive muscle states, only approximate ones).

Our method, then, reduces to choosing functions in (2) that best fit the averaged data before, during, and after enough trials by each subject (hundreds!) to enable him to achieve maximum speed. The resulting functions should tell us some valuable quantitative things about the muscle properties involved.

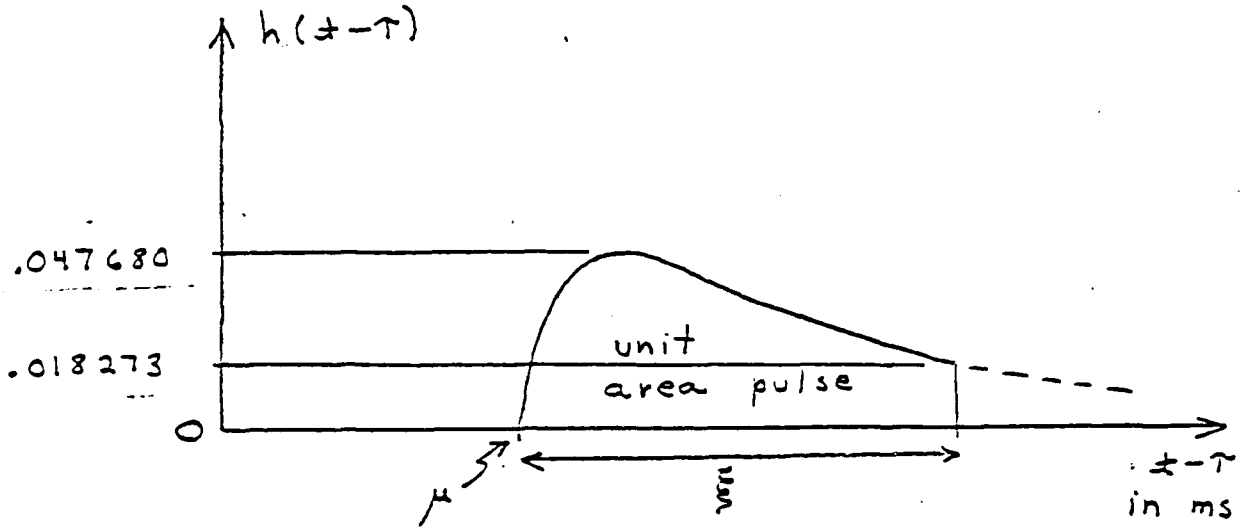
At the last level of analytical refinement, we let

$$\Psi_{IF}(t) = A_F \cos\phi(t) \int_{t-\mu-\xi}^{t-\mu} k_F \overline{EMG}_F(\tau) h_F(t-\tau)\, d\tau, \qquad (5)$$

where $A_F\cos\phi(t)$ converts flexor force to torque according to the geometry of Fig. II-1; $\overline{EMG}_F(\tau)$ is the smoothed EMG_F envelope at time τ; k_F is a constant of proportionality that we assume converts $EMG_F(\tau)$ into flexor force at τ (Messier, et al., 1971); and $h_F(t-\tau)$ is the normalized flexor twitch response at time t to a unit $k_F\overline{EMG}_F$ impulse that arrived at time τ.

Our $h_F(t-\tau)$ is defined in Fig. II-2, where we equate $h_F(t-\tau)$ with its extensor counterpart (for want of knowledge to do otherwise. This could be incorrect by perhaps 10% or so, especially after many practice trials). The shape of our h function is a ξ-truncation (for economy in computing) of Dijkstra et al.'s 1973a) twitch response function for biceps brachii muscle. But whereas their $\beta_1 = .015$ and $\beta_2 = .15$, we began with $\beta_1 = .05$ and $\beta_2 = .3$. As we continue to fit the incoming data, our β's will doubtless change in the direction of Dijkstra et al.'s.

Equation (5) assumes linearity in the accumulation of twitch responses. This is probably somewhat incorrect for our movement. Yet Sakitt, 1980, makes the same linearity assumption for an arm movement similar to ours and



$\mu = 20$ ms

$\xi = 30$ ms

define $t^* \equiv (t - \tau) - \mu$ ms

then $h(t^*) = q_f \left(\exp(-\beta_1 t^*) - \exp(-\beta_2 t^*) \right)$

where $q_f = .0819403$

so that $\int_0^{\infty} h(t^*)\, dt^* = 1.0$

with $\beta_1 = .05$

$\beta_2 = .3$

Figure II-2. Definition of $n_p(t-\tau) = n_E(t-\tau)$ in equation (6). See text for explanation.

obtains good results. In the future, however, we plan to try letting $h(t-\tau)$ vary as a function of

$$\int_{t-\tau-v}^{t-\tau} \overline{EMG}(\sigma)e^{-\alpha(t-\tau-\sigma)}d\sigma, \tag{6}$$

the exponentially-weighted $\overline{EMG}$ input from $t-\tau$ back to $t-\tau-v$ in time. This seems like the most expeditious way to introduce an appropriate nonlinearity for the accumulation of twitch responses into equation (5).

Letting $A_F k_F = C_F$ in (5) we get

$$\psi_{IF}(t) = C_F \cos\phi(t) \int_{t-\mu-\xi}^{t-\mu} \overline{EMG}_F(\tau)h_F(t-\tau)d\tau \tag{7}$$

Defining ψ_{IE} similarly and neglecting c_E in (2) (as does Sakitt, 1981. cf. also Lestienne, 1979), our final refined version of equation (1) becomes

$$\ddot{\phi}(t) = \psi_V \cos\phi(t)\left[\frac{C_E}{J}\int_{t-\mu-\xi}^{t-\mu} \overline{EMG}_E(\tau)h_E(t-\tau)d\tau - \frac{C_F}{J}\int_{t-\mu-\xi}^{t-\mu} \overline{EMG}_F(\tau)h_F(t-\tau)d\tau\right] \tag{8}$$

with the constraining assumptions given above. To a first approximation we can obtain C_E and C_F in (8) by replacing $J\ddot{\theta}$ by the isometric torque found

first in the flexor and then in the extensor directions for each subject. There are various good ways of measuring each subject's J (e.g., Hatze, 1980, refers to one he has developed), but we have not yet selected one.

We now discuss how the $\overline{EMG}$ signals in (8) are obtained. Figure II-3 shows an example test record. The EMG_{av} signals are rectified and filtered raw EMG signals. EMG_{av} signals are derived in an on-line computer which "continuously" leaks off a fraction of the present integral of the rectified EMG signal. Denoting the latter EMG_r, $\widehat{EMG}$ in Figure II-3, to a good approximation, is given by

$$\widehat{EMG}(t) = \int_0^t EMG_r(\tau)e^{-\alpha(t-\tau)}d\tau \qquad (9)$$

Thus if $EMG_r(\tau) = K$, a constant, then

$$\widehat{EMG}(t) = \frac{K}{\alpha}[1 - e^{-\alpha t}] \rightarrow \frac{K}{\alpha} \qquad (10)$$

as t gets large. Also, if $EMG_r(\tau) = C\tau$, C a constant, then

$$\widehat{EMG}(t) = \frac{C}{\alpha}t - \frac{C}{\alpha^2}[1 - e^{-\alpha t}] \rightarrow \frac{K}{\alpha}t \qquad (11)$$

for small t. Finally, it is easily shown that if $EMG_r = K$ as in (10) for a long time and then EMG_r becomes 0 at t,

$$\frac{d\,\widehat{EMG}(t+\varepsilon)}{d\varepsilon} = -Ke^{-\alpha\varepsilon} \rightarrow -K \qquad (12)$$

for small ε. Comparing (10) and (12) gives us a way to determine α from appropriate data samples.

Equations (10) - (12) enable us to find easily the $\overline{EMG}(t)$ quantities in (8) from $\widehat{EMG}(t)$ records such as shown in Fig. II-3. For example, given the

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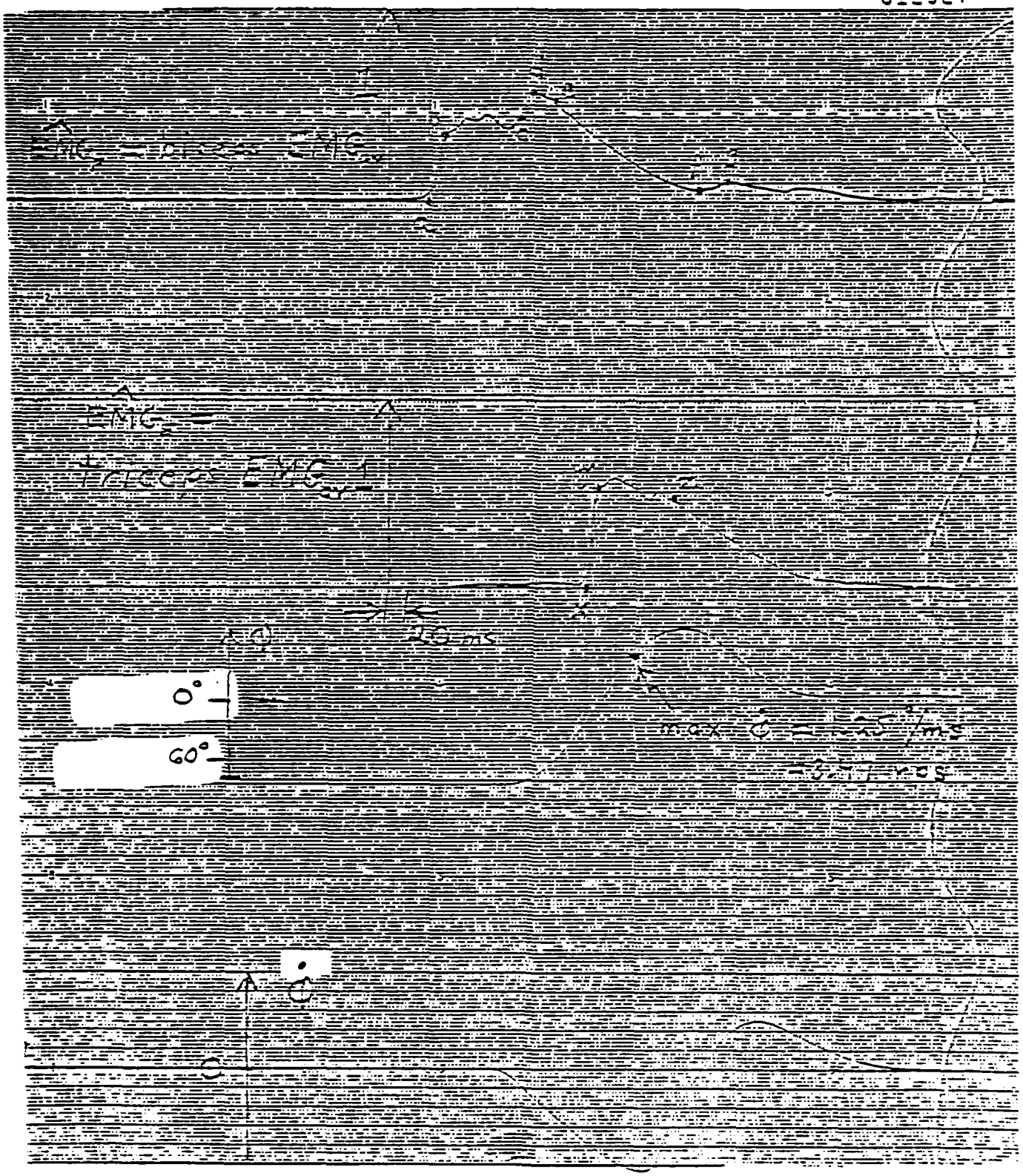

Figure II-3. An example test record. The EMG_{av} signals are marked to obtain $\overline{EMG}$ signals as explained in the text.

$\widehat{EMG}_F$ signal in Fig. II-3, we form a straight-line-segmental approximation to it by connecting a to b, b to c, etc. If in this approximation, a segment is essentially constant at K, we let $\overline{EMG}$ there equal $K\alpha$. If the segment rises with slope $C > 0$, we let $\overline{EMG}$ rise correspondingly with slope $C\alpha$. If the segment falls from K with slope -K, we let $\overline{EMG}$ there equal 0. Thus our $\overline{EMG}(t)$ equals α times the straight-line-segmental approximation to EMG except where that approximation falls off. The falloff intervals are easy to handle because the $\overline{EMG}$ signals for our movement are either on at high intensity or nearly off over most of every trial.

There is, however, one adjustment to the $\overline{EMG}(t)$ signal derived above that we need to make. In order to compensate for $\overline{EMG}$ inaccuracies during EMG falloff phases and intrinsic EMG distortions of nervous excitation to muscle at low signal levels, we shall augment our $\overline{EMG}$ signal with a $\theta(t)$ postural feedback term. This is justified by assumption 4 in the next section.

To make the adjustment, we denote the previous $\overline{EMG}$ by $\overline{EMG}^*$, and add a feedback term denoted $\overline{EMG}^{**}$. This gives us

$$\overline{EMG}(t) = \overline{EMG}^*(t) + \overline{EMG}^{**}(t) \tag{13}$$

on both the flexor and extensor sides. We define:

$$\overline{EMG}_F^{**}(t) = P_{F1}\left[\!\!\left[\theta(t-20) + P_{F2}\dot{\theta}(t-20) + P_{F3}\ddot{\theta}(t-20)\right]\!\!\right] G(t) \tag{14}$$

where the double-cusped brackets mean the same as in (4); where the quantity inside these brackets is $\theta(t)$ as predicted from θ, $\dot{\theta}$, and $\ddot{\theta}$ 20 ms earlier (20 ms being the total delay around the loop from γ receptor to α stimulation of muscle); where $G(t) = 0$ until θ first reaches 1 degrees, and $G(t) = 1$ thereafter; and where the P_{Fi} are constants (now set at $P_{F1} = .25$, $P_{F2} = 10$, $P_{F3} = 100$ -- recall that $\dot{\theta}$ is in degrees/ms and $\ddot{\theta}$ is in degrees/ms^2). Similarly,

$$\overline{EMG}_E^{**}(t) = P_{E1}\left[\!\!\left[-\left(\theta(t-20) + P_{E2}\dot{\theta}(t-20) + P_{E3}\ddot{\theta}(t-20)\right)\right]\!\!\right] G(t) \tag{15}$$

where $P_{Ei} = P_{Fi}$.

Finally, to keep (14) and (15) from causing everlasting ϕ oscillations, we change (1) to

$$J\ddot{\phi}(t) = Q_E(t) - Q_F(t) - C_\zeta\dot{\phi}(t) \qquad (16)$$

where the C_ζ viscoelastic damping factor (cf. Lestienne, 1979) is set "near" the level that just prevents a second ϕ overshoot of the 0 degree target position.

We now consider adaptation.

As our subjects accumulate more experience, we know from past results (Lagasse, 1975, e.g.) that their movement times will decrease. If the adaptations enabling this occur partly in the muscles, this should require us to modify the h functions in (8). Since in previous pilot experiments our flexor movement adapted several times faster and further than the opposite extensor movement, it could happen that only h_F in (8) adapts with practice. It also might develop that all adaptation occurs in the nervous system (cf. the com/cont/E model, next Section).

The crucial feature of our E/O/$\dot{\phi}$ model is that eventually it should locate and characterize the adaptive nonlinear effects involved when speed of movement is increased with practice. This should give us a mechanical perspective on how to train for speed, precision, grace and skill--in short, for well coordinated fitness.

We have included as Appendix A a listing of the PASCAL programme written by Valerie Congdon to compute $\dot{\phi}(t)$ and $\phi(t)$, as outlined above, using a file containing the $\overline{EMG}_F(t)$ and $\overline{EMG}_E(t)$ data for one movement. The program is well documented, hierarchically organized into procedural and functional levels identical to the analysis levels in this report, and specified so as to be easy to change. Ms. Congdon, who will leave UMass in January, 1981, has done an exemplary job.

2. The Com/Cont/E Model

This model concerns the functional organization of the neural control system for the $E/Q/\dot{\theta}$ model described above. It is an attempt to make precise only those conclusions that have been formulated by several experimenters as necessary consequences of their tests on fast arm movements involving the elbow and sometimes one other joint. The com/cont/E model is amenable to few significant neuroanatomical interpretations. Eventually we hope to obtain a good structural model that does not suffer from this defect, but that may be a couple of years away.

Our specification of the com/cont/E model arose out of the following Assumptions

1. For about the first 100 ms at least, our fast arm movement is entirely feedforward (is independent of any feedback), and thus is unaffected by stretch reflex or tendon afferents (cf. Wadman, et al., 1979; Desmedt and Godaux, 1978).
2. Selecting the levels L_F and L_E of flexor and extensor EMG activity require the first volitional decisions. (As noted by Kroll above, all of the flexor muscles act approximately in temporal and tensional unison with the biceps during our arm movement. Likewise for the triceps and the extensor muscles.) These levels are selected to set the speed and amplitude, M, of the movement with allowances for expected inertial loading of the limb and estimated fatigue of the muscles being activated (cf. Lestienne, et al., 1979; Lestienne, 1979; Wadman, et al., 1979; Wadman, et al., 1980).
3. The next movement decision is to set the relative timing of the first flexor and extensor EMG bursts. This determines the movement's combina-

tion of magnitude and speed, again allowing for the expected loading and muscle fatigue (cf. references in 2 above). Force-velocity and force-length effects are probably also included in this reckoning (Thorstensson, et al., 1976; Perrine and Edgerton, 1978), as well as viscoelastic braking (Lestienne, 1979; Maughan and Godt, 1979).

4. The flexor and extensor muscle tension levels at the end of the movement epoch when the postural maintenance phase is entered are variable, but the ratio R between these two levels, (as reflected by their leaky-integrated EMG levels) is a fixed postural command, and is not affected by the starting point, direction, amplitude, or speed of movement (Lestienne, et al., 1979; Sakitt, 1980). Presumably the EMG levels themselves at the end of the movement epoch are quickly adjusted if need be (how fast?) to obtain the desired postural stretch reflex stiffness (Houk, 1976). Since the biceps tendon reflex loop delay is about 35 ms, the stretch reflex loop delay is about 50 ms (Marsden, et al., 1976), and our actual arm movement duration is about 300 ms, the presently unknown speed with which the set point of the stretch reflex is reset and the reflex subsequently engaged will be imortant to discover.
5. In males at least, continued practice of a fast arm movement reduces execution time and alters the movement's EMG signature (Lagasse, 1975).

In the light of the above assumptions, we now give a point-by-point description of the com/cont/E model (Figure II-4):

1. At the top of Figure II-4, R, L, M, ILF, and FF are commands issued from the volitional decision center for our fast arm movement (this "center" might be distributed over a large part of the brain). R is the ratio described in assumption 4 above; L times g_p, $L.g_p$, and $L.g_e$ are the

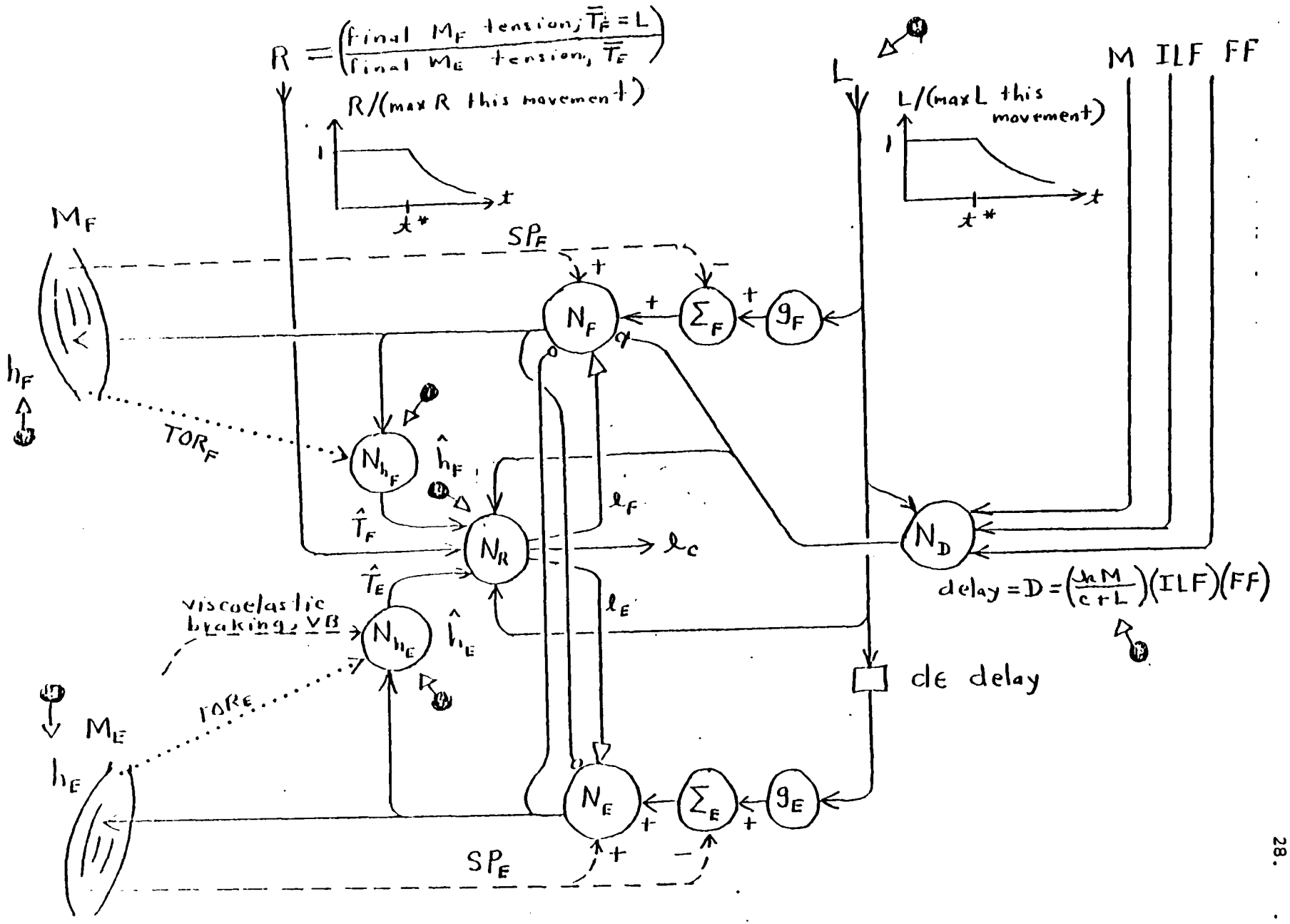

Fig. II-4. Functional organization of controller of volitional fast arm movement. See text for explanation.

levels L_F and L_E described in 2 above; M is defined in 2; ILF, the inertial loading factor, and FF, the fatigue factor, referred to in 2 and 3 are appropriately scaled for multiplication into the subject's moment of inertia, J, for the arm movement in question. Thus ILF $\geq$ 1, with IFL = 1 when no external load is added; and $0 < FF \leq 1$, with FF = 0 interpreted as complete paralysis and FF = 1 as zero fatigue (defined in terms of force development capability).

2. N_F and N_E are loosely interpretable respectively as flexor and extensor motorneuronal pools that drive either the flexor and extensor arm musculature for our movement or just the biceps and triceps.
3. At t = 0 the flexor EMG is activated. t* is the time at the end of the first extensor EMG burst. The time courses of R and L with respect to t* are hypothesized as shown at the top of the figure.
4. At t = 0, L turns N_R on, which then turns N_F on (see 6 below), all with delay less than $d\varepsilon$. N_f's output firing rate rises and falls somewhat sporadically, but peaks at the $L.g_F$ level. The stretch reflex pathway, SP_F, into N_F is intially inactive as noted in assumption 1 above. N_F's output equals the algebraic sum of its inputs. N_F keeps N_E shut off over the reciprocal inhibitory pathway.
5. At time D, a shot out of N_D turns N_F off to end the first flexor EMG burst. c and k in the formula for D are scaling constants. Since L turns N_D on, the value of c would be 0 if the flexor muscle M_F were perfectly linear in L and if no co-contraction of M_F and M_E were necessary to stabilize the elbow joint during movement. Since neither of these conditions holds, $c > 0$. After N_F shuts off, N_E is no longer inhibited and turns on. Then N_E keeps N_F shut off by reciprocal inhibition.

6. Backtracking a bit, only one of N_R's outputs is on at a time. Initially, L turns N_R's ι_F output on, which is necessary to keep N_F on. Later N_D's output shot switches N_R's output over to ι_E, which is necessary to keep N_E on.

7. N_R's input T_F provides N_R with an estimate of M_F's tension, as computed (or simulated) by Nh_F. The function h_F is Nh_F's estimate of M_F's twitch response function h_F (see Part II-A-1) on tne E/Q/ϕ model). In contrast with the nervous system, we would model Nh_F's output at time t with $\int_0^t N_F(o)\hat{h}_F(t-\sigma)d\sigma$. By assumption 4 above, the flexor tendon organ response, TOR_F, is effectively unavailable to Nh_F for at least 80-85 ms. Mutatis mutandis, N_R's extensor circuits are the same as its flexor ones.

8. If our movement is entirely feedforward, some such centers as Nh_F and Nh_E are necessary to control N_F and N_E's development of an appropriately stiff arm posture at the end of the movement (assumption 4 above). The possibility that there are Nh_F and Nh_E circuits in the cerebellum seems to fit Eccles' concept of the cerebellum as a control reference in fast voluntary movements (Eccles, 1977). Lagasse, 1975's results showing that EMG temporal organizations for fast human limb movements don't change with fatigue support the idea that such movements are entirely feedforward.

9. When N_R's $\hat{T}_E$ input reaches the desired final level, N_R's output switches from ι_E to ι_F in case $\hat{T}_E$ is not up to the desired final level, and to ι_C in case $\hat{T}$ is up to its final level. An active ι_C signals the end of the movement and subsequent reinstatement of the normal postural mainte-

nance mechanisms. The desired final flexor and extensor tensions are denoted $\overline{T}_F$ and $\overline{T}_E$, and are assumed to satisfy $\overline{T}_E R = \overline{T}_F = L$ (assumption 4 above). Thus $(\overline{T}_F/\overline{T}_E) = R$, and the N_R component in Fig. II-4 has sufficient inputs to compute all of its outputs.

10. If we assume some background SP_F, SP_E, and other noise, the sequence of events in 9 will not always be as simple as described. The first N_F or N_E EMG burst might sometimes be cut off prematurely, forcing another one or even two small bursts later on (see the L time course, top right in Figure II-4). The famous third EMG burst (e.g., Wadman, et al., 1980) might be accounted for in this way -- an attractive hypothesis because Kroll's experiments to date with highly practiced subjects usually don't show a third EMG burst. The ability of the control components in Fig II-4 to learn (with lots of practice) the amount of viscoelastic braking that actually occurs, the force-velocity and force-length relationships of the muscles, and various nonlinearities of the system, offer other adaptation possibilities.

11. The ●→ symbol in Fig. II-4 points to likely adaptation sites as the same movement is practiced over successive trials and days.

12. N_D and the (N_D, Nh_F,Nh_E) complex could be combined instead of functionally partitioned as shown, but that would defeat the purpose of the com/cont/E model.

II-8 Proposed Modelling Studies Next Year

We wish to proceed on three fronts:

1. The $E/Q/\dot{\phi}$ model will be adjusted, and perhaps revised or augmented, to fit Kroll's experimental results. His tests with different loads and fatigue levels should be very helpful to us during this phase.
2. The Com/Cont/E model will be scrutinized. In particular, the N_D and N_h components will be checked for plausibility against Kroll's fatigue and load variational test data. We now believe that "efferent-copy" M_F simulators such as the N_h functions actually exist (perhaps at the highly integrative thalamic level atop the brain stem reticular formation, or in the cerebellum), but we shall seek more evidence to support this idea. We also plan pilot tests of various kinds to further elucidate the role of stretch reflexes at different times during and immediately following fast arm movements. To date, Wadman et al. (1979) have shown that when a subject's planned fast arm movement is unexpectedly blocked, the pattern of EMG activity over at least the first 100 ms is the same as before blocking. But we know of nothing else in the literature on the dynamics of rapidly changing the set points of stretch reflexes.
3. We shall develop computer programs in PASCAL for solving the nonlinear delay-differential equations that will arise when the $E/Q/\dot{\phi}$ model is augmented to include feedback stretch reflex information. Banks (1979, 1980) has recently developed the requisite mathematical methods, but as yet PASCAL programs to implement them do not exist.

In 1 to 3 above, we shall be especially watchful of adaptation parameters and characteristics. In year three of the contract, we shall apply

the results of the first two years to a search for better ways to measure and train optimally coordinated fast human limb movements, with an emphasis on kinesthetic learning.

NOTE: Next page is 55.

REFERENCES

Bahill, A. T. and L. Stark. The trajectories of saccadic eye movements. Scientific American 241:108-117, 1979.

Bahill, A. T., F. H. Hsu, and L. Stark. Glissadic overshoots are due to pulse width errors. Archives of Neurology 35:138-142, 1978.

Bailey, G.B. and R. Presgrave. Basic Motion Time Study. New York: McGraw Hill, 1958.

Barnett, C. H. and D. Harding. The activity of antagonist muscles during voluntary movement. Annals of Physical Medicine 2:290-293, 1955.

Barto, A. and R. Sutton. Technical report in the process of approval on AF contract no. F33615-77-C-1191 at the University of Massachusetts-Amherst, Computer Science Department entitled: Adaptive network simulations, fall, 1979.

Beaunis, H. Recherches physiologiques sur la contraction simultanee des muscles antagonistes. Archives de Physiologie Normale et Pathologique 1:110-131, 1889.

Bell, C. Anatomy and physiology of the human body. Philosophical Transactions 7:110-151, 1823.

Bierman, W. and H. J. Ralston. Electromyographic study during passive and active flexion and extension of the knee of the normal human subject. Archives of Physical Medicine and Rehabilitation 46:71-75, 1965.

Burghes, D. and A. Downs. Modern Introduction to Classical Mechanics and Control. New York: Halstead, 1975.

Clark, M. and L. Stark. Time optimal behavior of human saccadic eye movement. IEEE Transactions on Automatic Control 20:345-348, 1975.

Clarke, D. H. Correlation between the strength/mass ratio and the speed of an arm movement. Research Quarterly 31:570-574, 1960.

Corser, T. Temporal discrepancies in the electromyographic study of rapid movement. Ergonomics 17:389-400, 1974.

Demeny, C. Du role mecanique des muscles antagonistes. Archives de Physiologie Normale et Pathologique 2:747-752, 1890.

Descartes, R. L'Homme. Paris: Louis de la Forge, 1677.

Desmedt, J. and E. Godaux. Voluntary motor commands in human ballistic movements. Annals of Neurology 5:415-421, 1979.

Dijkstra, Sj., J. J. Denier van der Gon, T. Blange, J. M. Karemaker, and A. E. J. L. Kramer. Simplified sliding-filament model for simulation purposes. Kybernetik 12:94-101, 1973.

Dijkstra, Sj. and J. J. Denier van der Gon. An analog computer study of fast, isolated movements. Kybernetik 12:102-109, 1973.

Golla, F. and J. Hettwer. A study of the electromyograms of voluntary movement. Brain 47:57-69, 1924.

Granit, R. The Purposive Brain. Cambridge: MIT Press, 1977.

Henry, F. M. Factorial structure of speed and static strength in a lateral arm movement. Research Quarterly 31:440-448, 1960.

Henry, F. M. Factorial structure of individual differences in limb speed, reaction, and strength. Research Quarterly 33:70-74, 1962.

Henry, F. M., and J. D. Whitley. Relationships between individual differences in strength, speed and mass in arm movement. Research Quarterly 31:24-33, 1960.

Houk, J. C. An assessment of stretch reflex function. Brain Research 44: 303-314, 1976.

Hudgins, C. V. The incidence of muscular contraction in reciprocal movements under conditions of changing loads. Journal of General Psychology 20:327-338, 1939.

Kilmer, W. and Probert. Depletion models for ecosystems with continuously delayed resource renewals. Mathematical Biosciences (in press).

Kots, Ya. M. The organization of voluntary movement: neurophysiological mechanisms. New York: Plenum Press, 1977.

Kroll, W. Isometric cross transfer effects under conditions of central facilitation. Journal of Applied Physiology 20:297-300, 1965.

Kroll, W. Central facilitation in bilateral vs. unilateral isometric contractions. American Journal of Physical Medicine 44:218-223, 1965.

Kroll, W. Isometric fatigue curves under varied intertrial recuperation periods. Research Quarterly 39:106-115, 1968.

Kroll, W. Effects of local muscular fatigue due to isotonic and isometric exercise upon fractionated reaction time components. Journal of Motor Behavior 5:81-94, 1973.

Kroll, W. Fractionated reaction and reflex time before and after fatiguing isotonic exercise. Medicine and Science in Sports 6:260-266, 1974.

Kroll, W. and P. M. Clarkson. Age, isometric knee extension strength, and fractionated resisted response time. Experimental Aging Research 4: 389-409, 1978.

Lagasse, P. P. The prediction of maximum speed of human movement by two selected muscular coordination mechanisms and by maximum static strength and the effects of practice and fatigue upon maximum speed of human movement mechanisms. Unpublished Ph.D. dissertation, University of Massachusetts, Amherst, 1975.

McMahon, T. A. and P. R. Greene. Fast running tracks. Scientific American 240:148-163, 1978.

Matthews, P. B. C. Mammalian muscle receptors and their central actions. London: Arnold, 1972.

Miles, F. A. and E. V. Evarts. Concepts of motor organization. Annual Review of Physiology 30:327-362, 1979.

Nelson, R. C. and R. A. Fahrney. Relationship between strength and speed of elbow flexion. Research Quarterly 36:455-463, 1965.

Patton, N. J. and O. A. Mortensen. An electromyographic study of reciprocal activity of muscles. Anatomical Records 170:255-268, 1971.

Person, R. S. An electromyographic investigation on co-ordination of the activity of antagonist muscles in man during the development of the motor habit. Pavlovian Journal of Higher Nervous Activity 8:15-25, 1958.

Pettigrew, J. Lectures on the physiology of the circulation in plants, in lower animals and in man. In: Tilney, F. and H. F. Pike. Muscular coordination experimentally studied in relation to the cerebellum. Archives of Neurology and Psychiatry 13:289-334, 1925.

Phillips, C. G. and R. Porter. Corticospinal neurones: their role in movement. New York: Academic Press, 1977.

Rasch, P. J. Relationship of arm strength, weight, and length to speed of arm movement. Research Quarterly 25:328-332, 1963.

Sherrington, C. S. Integrative Action of the Nervous System. New York: Yale University Press, 1906.

Smith, L. E. Individual differences in strength, reaction latency, mass and length of limbs, and their relation to maximal speed of movement. Research Quarterly 32:208-220, 1961a.

Smith, L. E. Relationship between explosive leg strength and performance in the vertical jump. Research Quarterly 32:405-408, 1961b.

Smith, L. E. Specificity of individual differences of relationship between strengths and speed of forearm flexion. Research Quarterly 40:191-197, 1969.

Stelmach, G. E. Motor Control: issues and trends. New York: Academic Press, 1976.

Stetson, R. H. and H. D. Bouman. The coordination of simple skilled movements. Archives Neerlandaises de Physiologie de l'homme et des Animaux 20: 177-254, 1935.

Tilney, F. and F. H. Pike. Muscular coordination experimentally studied in its relation to the cerebellum. Archives of Neurology and Psychiatry 13:289-334, 1925.

Wilkie, D. R. The relation between force and velocity in human muscle. Journal of Physiology (London) 110:249-280, 1950.

APPENDIX A

Article: An EMG-Level Muscle Model for a Fast Arm Movement to Target

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An EMG-Level Muscle Model for a Fast Arm Movement to Target*

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ABSTRACT

A model of human muscle action is presented for a maximally fast, large-amplitude forearm movement to target. The inputs to the model are approximately the biceps and triceps EMG envelopes over a single movement. The model's output gives the corresponding displacement angle of the forearm about a fixed elbow position as a function of time. The idea of the model is to conceive of both EMG input drives as successions of millisecond input pulses, with each pulse resulting in a muscle tension twitch. Every twitch is amplitude-scaled, parametrically-shaped, and duration-limited as a function of the muscle's contractile history thus far in the movement. The muscle tension at any time t is the sum of the residual tension levels of all twitches begun before t.

The model was developed and tested with special reference to two subjects: one, according to the model dynamics, was a comparatively slow-twitch type, and the other modelled as a fast-twitch type. Good agreement was found between model output and subject response data whenever the subject's EMG's were "synchronous." The model can be used to characterize each subject's responses by a suite of twitch characteristics. This will enable us to check the accepted but now suspect correlation between muscle biopsy- and performance-determined muscle twitch type.

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1. INTRODUCTION

We present a mathematical model of the elbow flexor (biceps group) and extensor (triceps) muscle actions for the human arm movement outlined in Fig. 1. The inputs to the model are the $\overline{EMG}$ signals referred to in the caption of Fig. 2. They approximate the envelopes of raw EMG signals, are are derived from EMG signals such as shown in Fig. 2a) and b) by the method described in Kilmer, et. al. (1981). The output of the model corresponding to one trial of the type in Fig. 2 is a calculation of an arm displacement function such as shown in Fig. 2c).

This is the first attempt we know of to analyze EMG(t)-to-$\mathcal{T}$(t) transformations where $\mathcal{T}$ is the tension noted in Fig. 1) in muscles controlling fast, large-amplitude limb movements to a target. The most closely related modelling works to date are: Stein and Oguztoreli (1981)'s small signal linear analysis of the mammalian-neuromuscular and reflex system; Hatze's (1978, 1980, 1981) papers at a more detailed level involving state equations for numbers of motor units in different contractile states; Sakitt's (1980) steady-state spring model for postural changes similar to that shown in Fig. 1; and Dijkstra et. al.'s (1973a, 1973b) original cross-bridge model and analog computer simulation of small amplitude human forearm movements.

Our model attempts to better determine the correlations between: 1) the speed (power) and shape of maximally fast, large-amplitude human arm movements to a target; 2) the proportions of fast- vs. slow-twitch fibers, as determined by muscle biopsy in subjects making such movements; and 3) the physiologically effective twitch speeds involved at different stages of each such movement when performed by highly skilled (i.e., well-practiced) subjects. As discussed below, some evidence has accumulated to suggest that

the fast- vs. slow-twitch dogma of mammalian skeletal muscle action may be inaccurate to some degree.

Several investigators have experimented with human movements that differ from that of Fig. 1 primarily in that they are performed horizontally so as to eliminate the changing effects of gravity. Most notable among them are Wadman, et. al. (1979, 1980a, 1980b), Lestienne et. al. (1979), and Lestienne (1979). Lagasse (1975), and Kroll and associates (e.g. Kroll (1974)), however, have collected the most suitable data for our purposes by using essentially the Fig. 1 paradigm on subjects whose EMG signatures were always cleaned, sharpened, and stabilized through extensive pre-test practice. We chose the resulting so-called "synchronous" EMG's to analyze with our model. (We note below that the confounding effect of gravity in Fig. 1 is apparently negligible.)

Kroll's protocol for each of his 29 subjects (about half men and half women, all right-handed) involved four practice days with 50 trials per day, followed by five exercise (i.e. test) days with 20 trials to establish baseline and 5 test trials--all for each of three load levels. We derived all our model inputs from trials using the smallest added load, namely three times the arm's moment of inertia for the movement. This gave us signal plots of about the right size on all channels for the modelling accuracy we sought. All subjects were told to "make the movement as fast as you can, and stop at the 0° target position." The emphasis was on speed of movement, not targeting precision. Under these circumstances, the published data on more or less fast movements of other human body parts (eyes, head, hands, fingers, legs) are often at variance with our results.

We selected trial data from two subjects, Hintr and Chan, both 25-year old males, to help explain our models. In the next Section, we present the conceptual organization of our basic model, the U-Model, pictorially, leaving almost all of the mathematical formulae till later. Then, following a mathematical specification of U-Model, we show how it is augmented to J-Model to fit the data of Chan's much faster trials. We conclude with some possible applications of J-Model.

2. CONCEPTUAL MODEL

The purpose of our models is to better understand, in a quantitative way, how nervous drives to muscles as represented by their EMG's lead to schedules of muscle tension and thence to movements of the kind outlined in Fig. 1. The U-Model, and its J-Model augmentation, are embodied in a PASCAL program, called FASMOV, that accepts piecewise-linear approximations of biceps and triceps EMG waves taken from a dynograph recorder, and produces a prediction of the subject's corresponding arm movement as a function of time. Kilmer, et. al. (1981) specifies how FASMOV computes effective nervous drives, $\overline{EMG}_F$ and $\overline{EMG}_E$, from flexor and extensor dynograph EMG outputs, $\widehat{EMG}_F$ and $\widehat{EMG}_E$, respectively.

Fig. 3 gives a graphic outline of the main concepts employed in the U-Model. At the top of the Figure we imagine a constant flexor $\overline{EMG}_F(t)$ drive that lasts for 100 ms. We conceive of this drive as a succession of 100 contiguous 1 ms-pulses, each of amplitude $\overline{EMG}_F$ (thus there should be about ten times as many vertical lines as shown in $\overline{EMG}_F(t)$). We assume that each millisecond $\overline{EMG}_F$ pulse produces an ensuing twitch of tension whose time course is like that of a small wave in the baseline train of Fig. 3b), and whose area is a scale factor times the $\overline{EMG}_F$ value (Meissier, et al., 1971). The shape of the leading twitch in the Fig. 3b) train is

essentially that used by Dijkstra et al. (1973a). We denote the electro-mechanical delay between the time of an $\overline{EMG}_F$ pulse and when the corresponding twitch starts by μ_F. A value of 60 ms for μ_F has given us a best fit to the data in all our models so far. This value is midway between the 73 ms and 76 ms values found experimentally for biceps by Lagasse (1975) and by Ward (1978) respectively, and the 55 ms and 52 ms values found experimentally for triceps by Lagasse (1975) and by Morris and Beaudet (1980) respectively.

In Fig. 3b) our twitch durations range from 60 ms at the outset to 90 ms at the end. These durations are generally compatible with those measured, e.g., in human thumb flexor muscles by Gydikov, et al., 1976. Their fast fatiguable motor unit twitch times ranged from 91 to 125 ms before fatigue and from 125-333 ms after. We do not suppose that our 90 ms terminal twitch time is a "fatigued" duration; rather we refer to it as a "saturated" duration. Note that we clipped our saturated twitch durations off prematurely as shown in Fig. 3b); this was only to save computer memory during simulation runs.

Letting ξ_F denote flexor twitch duration, we assume that a millisecond $\overline{EMG}_F$ pulse at time t causes a tension twitch given by

$$h_F(\tau) = \begin{cases} q_F(\exp(-\beta_{1F}(\tau-\mu_F)) - \rho\exp(-\beta_{2F}(\tau-\mu_F))) \\ \qquad \cdot \qquad \text{for } t+\mu_F \le \tau \le t+\mu_F+\xi_F \\ 0 \text{ for all other } \tau \end{cases} \tag{2.1}$$

where $\rho = 0$ until the first adjustment of twitch shape in the Fig. 3b) train, and $\rho = 1$ thereafter.

Except for the ρ factor, (2.1) is Dijkstra's (1973a) formula. Our β_{2F} equals their $\beta_2 = 0.15$, but whereas their $\beta_1 = 0.015$, we allowed our β_{1F} to range from 0.05 initially to 0.01 at saturation in both our models. For every different β_{1F} value we recalculated q_F in (2.1) so as to renormalize the area under $h_F(\tau)$ to nearly 1.0 (details in Kilmer et al. (1981)).

Fig. 3b) depicts a gradual change in the $h_F(\cdot)$ twitch shape, which was our original idea. But to help cut FASMOV's run costs we programmed FASMOV to change $h_F(\cdot)$ shapes less gradually (i.e., less often). Because q_F (among other things) has to be changed each time $h_F(\cdot)$ changes, we refer to the entire set of $h_F(\cdot)$- or $h_E(\cdot)$-changing operations in FASMOV as a "reque". Fig. 4 shows all but the last $h_F(\cdot)$ and $h_E(\cdot)$ that were used during the Hintr1 run, where only nine reques in all occurred (the details on when and how to reque appear in Kilmer et al. (1981)).

The total flexor tension wave $\mathcal{T}_F(t)$ in Fig. 3b) is found by adding up at each time all of the $h_F(\tau)$ wave values that are not zero at t. These nonzero values would be the $h_F(\tau)$ values caused by $\overline{EMG}_F$ pulses occurring between the times $(t - (\mu_F + \xi_F))$ and $(t - \mu_F)$. ξ_F here is a twitch duration such that the oldest nonzero twitch at t rose from zero at time $t - \xi_F$ and ξ_F was the twitch duration in effect then (cf. next Section). Our method of producing the $\mathcal{T}_F(t)$ wave assumes linearity in the accumulation of twitch effects. This is probably somewhat incorrect, but Sakitt, 1980, e.g., made the same assumption for an arm movement similar to ours, and obtained good results. The simplification was justified when the model outputs agreed very well with the subject's data.

On the right end of Fig. 3b) we show the $\mathcal{T}_F(t)$ wave jumping up from its sum-of-$h(\tau)$'s value, given by the dotted curve, to an amplitude about three times greater. Let S_R denote the multiplication factor in this jump, which occurs when $\dot{\ell}$ changes from negative to positive, i.e. when the biceps stops contracting and starts stretching. Heckathorne and Childress (1981) show curves for slowly lengthening and contracting rates in human biceps that are strikingly compatible with the S_R values for which our models best fit the subjects' data: $S_R = 3.0$ in all Hintr runs, and $S_R = 2.5$ in all Chan runs. We use the

same S_R value for both triceps and biceps in each model simulation run; but since stretching one of these muscles means contracting the other, a jump of $\mathcal{T}_F(t)$ to $S_R\mathcal{T}_F(t)$ is always accompanied by a jump of $\mathcal{T}_E(t)$ to $\mathcal{T}_E(t)/S_R$, and vice versa.

Fig. 3c) outlines our last major concept. As the integral of $\mathcal{T}_F(t)$ grows, the muscle becomes less efficient in converting $\overline{EMG}(t)$ drive into tension. We represent this growing inefficiency by a multiplying factor for $\mathcal{T}_F(t)$, which we denote by $\exp(-\sigma_F^*)\cdot\exp(-\eta_F^\circ)$--the rationale appears below. The continuous curve in Fig. 3c) represents a typical $\exp(-\sigma_F^*)$ during a movement. The quantity $-\sigma_F^*(t)$ is just a scaled leaky integral of $\mathcal{T}_F(\tau)$ from $\tau=0$ to $\tau=t$. The dotted adjustment to $\exp(-\sigma_F^*)$ in Fig. 3c) is due to the $\exp(-\eta_F^\circ)$ factor. Initially η_F is nonzero (ranging from -0.5 to -1.8 in simulation runs so far); this quantity represents a "primed" tension-producing state of the muscle at tension onset. At the first reque of each simulation run, $\dot{\eta}_F$ is reset to zero for the remainder of the run (in the U-Model, but there is an extra twist in the J-Model). The reset represents a loss of prime in the muscle state.

To see the rationale for Fig. 3, we must sketch a bit of muscle physiology (cf. e.g., Stein, 1980):

> Nervous action potentials into a muscle cell cause the release of calcium ions from the endoplasmic reticulum. These ions diffuse to sites on actin filaments and thereby enable myosin heads to lock onto the filaments, pulling them toward the center of the sarcomere. Each myosin head can repeat this action by locking sequentially onto a series of sites along an actin molecule. Between each successive pair of such head attachments, a biochemical sequence of three major actomyosin molecular states must be traversed (Eisenberg, et al., 1980). R. Cooke (in private correspondence in 1979) and others have guessed that this three-state cycle may last on the order of 50 to 100 ms. Thus, during a movement such as ours, lasting three or four hundred milliseconds, more and more of the most accessible (and perhaps most effective) attachment sites pass into restorative states that cannot be influenced by calcium ions to produce force increments. As our movement progresses, then, smaller and smaller fractions of newly freed calcium ions yield force increments before gathering again in the reticulum. Moreover, the diffusion times of those ions that do generate forces presumably increase, making composite muscle twitches caused by later-arriving nervous action potentials last longer.

We represent part of these sketched effects in Fig. 3b) by moving to longer twitch amplitudes for later-starting twitches (each twitch area being normalized to nearly 1.0). The rest of the saturation effect is represented in Fig. 3c) by the multiplier $\exp(-\sigma_F^*)$. (The priming factor $\exp(-\mathring{n}_F)$, though not yet mechanically interpretable, is necessary for the models to fit the subjects' data.) $\sigma_F^*(t)$ is determined by

$$\frac{d\sigma_F^*(t)}{dt} = -(1-\delta_F)\sigma_F^*(t) + \gamma_F Q_F(t), \qquad (2.2)$$

where $(1-\delta_F)$ (typically about 0.015) is a rate constant for recovery of σ_F^* toward zero, $Q_F(t)$ is flexor torque (approximately equal to $T_F(t)$ after the initial stage of the movement), and γ_F (typically about 1.5) is a rate constant for the growth of $\sigma_F^*(t)$ as a function of $Q_F(t)$ ($Q_F(t)$ maxima vary from 0.0172 in HintrA to 0.0342 in Chan2). The $\sigma_F^*(t)$ function in (2.2) solves to

$$\sigma_F^*(t) = \int_0^t \gamma_F Q_F(\tau) e^{-(1-\delta_F)(t-\tau)} d\tau \qquad (2.3)$$

since $\sigma^*(0) = 0$.

This function is the simplest one that exhibits the desired shape and that also gives good modelling results.

Fig. 3 contains all flexor terminaology, but everything there applies also to the extensor except that $\mathring{n}_E = 0$; the extensor prime is dissipated before the onset of the big extensor burst because the extensor must first stabilize the elbow joint for the flexor to pull against.

3. THE U-MODEL

We now give a top-down description of our basic model, the U-Model. All of its parameter values apply uniformly (hence the "U") over the entire duration of each run. In the J-Model, which is simply an augmented U-Model, some parameter values are adjusted during each run (cf. the Fig. 12 caption).

FASMOV runs as U-Model when the two parameters T_{DE} and T_{DF} are set to values exceeding the trial time for the run in question. Each run of FASMOV simulates one movement, or trial, of a subject.

Fig. 1 gives the elements of our model. Though simple, similar models have been employed by Messier, et al.(1971), Sakitt (1980), and others with excellent results. Refinements of the Fig. 1 scheme lead invariably to distributed parameter systems that are very difficult to analyze in the terms we have chosen, so we have not considered them.

We ignored the effects of gravity in our equations. This is justified by the high quality of our results as discussed below.

The top-level analysis formula for our movement is

$$J\ddot{\phi}(t) = Q_E(t) - Q_F(t) - C_\xi\dot{\phi}(t), \qquad (3.1)$$

where each dot over ϕ indicates a time derivative, Q_F and Q_E are the respective torques about the elbow as exerted by the flexor muscle group and the extensor muscle, and $C_\xi\dot{\phi}$ is a viscoelastic damping torque (cf. Lestienne, 1979). Since the flexor group acts synergistically (cf. Lagasse, 1975, e.g.), we shall regard it as synonymous with the biceps. J is the arm's moment of inertia for rotations, (over a ϕ change from 60° to about -30°) about a fixed elbow joint as shown in Fig. 1. In our models we normalized J to 1.0.

At the second level of analysis, we let

$$Q_e(t) = \psi_{VE}(\dot{\phi}(t)) \cdot \psi_{IE}(t) \qquad (3.2)$$

ψ_{IE} in (3.2) may be regarded as the maximum (over $-90° < \phi \leq 90°$) isometric torque producible by the extensor, given the smoothed extensor EMG envelope, $\overline{EMG}_E$, over the recent past. ψ_{VE} is roughly interpretable as a force-velocity factor. We got best fit to our data with ψ_{VE} values slightly less than 1.0 in all cases. This is not incompatible with the force-velocity relations obtained by Thorstensson et al. (1976) and Perrine and Edgerton (1978) in their

studies of the human knee, nor with Lagasse's (1975) work on fast human forearm movements (quite like ours only untargeted). Ultimately, however, ψ_{VE} must be regarded as a successful coverup of the following considerations: Maton and Bouisset (1977) found that the muscles of the biceps group all show a uniform level of activity during isometric contraction regardless of muscle length. Messier, et al. (1971) report that under static loads, "the averaged EMG is directly proportional to muscle tension and the constant of proportionality is independent of muscle length"; but Messier's tension levels were low. Zuniga and Simmone (1969) show that at higher tension levels the tension-EMG relationship saturates quadratically in tension. Certainly tension production in muscles that are rapidly stretching or contracting is very different from that in muscles that are bearing static loads. During changes of muscle length, both passive (visco) elastic and active molecular force components arise.

For the flexor side we let

$$Q_F(t) = \psi_{VF}(\dot{\phi}(t)) \cdot \psi_{IF}(t) \tag{3.3}$$

where ψ_{IF} is similar to ψ_{IE} and $\psi_{VF} = \psi_{VE}$. Equating ψ_{VE} to ψ_{VF} for our movement is probably slightly inaccurate because flexor and extensor muscle length change in opposite directions and when one is most actively driven the other is responding most passively (the joint must be stabilized, so there are never any purely passive muscle responses, only approximately passive ones).

At the third level of analysis we let

$$\psi_{IF}(t) = \mathcal{A}_F \cos \phi(t) \int_{t-\mu_F-\xi_F}^{t-\mu_F} \mathcal{K}_F \, \overline{EMG}_F(\tau) \mathcal{H}_F(t-\tau) \, d\tau \tag{3.4}$$

The integral in (3.4) is flexor tension at time t, $\mathcal{T}_F(t)$, and the $\mathcal{A}_F \cos \phi(t)$ factor in (3.4) converts $\mathcal{T}_F(t)$ into torque. Initially in each FASMOV run, $\mathcal{A}_F = A_F$, where A_F is the length of the effective flexor moment arm as shown in Fig. 1. Whenever $\dot{\phi}(t)$ changes from negative to positive during a run,

$\mathcal{A}_F$ switches from A_F to $A_F S_R$ as discussed in the previous Section. Whenever $\dot{\phi}(t)$ changes back from greater than zero to negative, as it sometimes does at late stages in a run, $\mathcal{A}_F$ switches back from $A_F S_R$ to A_F.

Referring to the Hintrl run, $\mathcal{H}_F(t-\tau)$ in (3.4) is that $h_F(\cdot)$ curve in Fig. 4 that was in effect when the muscle twitch corresponding to the hypothetical $\overline{EMG}(\tau)$ pulse started up from zero. This startup time was $\tau + \mu_F$, where μ_F is the flexor electromechanical coupling time (i.e., the time between a nervous stimulus to a muscle and the beginning of the corresponding tension twitch; cf. Norman and Komi (1979)). Thus if $t-\tau < \mu_F$, $\mathcal{H}_F(t-\tau)$ is zero because the tension twitch for $\overline{EMG}_F(\tau)$ has not yet begun. Letting ξ_F denote the duration of a tension twitch starting up from zero at $\tau + \mu_F$, if $t-\tau > \mu_F + \xi_F$, $\mathcal{H}_F(t-\tau)$ is zero because the tension twitch for $\overline{EMG}_F(\tau)$ has already ended. These zero regions of $\mathcal{H}_F(\cdot)$ determine the upper and lower time limits of integration in (3.4).

$\mathcal{K}_F$ in (3.4) equals $K_F e^{-\sigma_F} = K_F e^{-\sigma_F^*} e^{-\eta_F}$, where K_F is a flexor EMG-to-tension scale factor, and $e^{-\sigma_F^*} e^{-\eta_F}$ is as explained in the previous Section in reference to Fig. 3.

For the extensor muscle, in place of (3.4) we have

$$\psi_{IE}(t) = \mathcal{A}_E \cos \phi(t) \int_{t-\mu_E-\xi_E}^{t-\mu_E} \mathcal{K}_E \, \overline{EMG}_E(\tau) \mathcal{H}_E(t-\tau) \, d\tau \qquad (3.5)$$

The explanation of (3.5) differs from that of (3.4) in only two respects: First, $\mathcal{K}_E = K_E e^{-\sigma_E}$, where $\sigma_E = \sigma_E^*$ with σ_E^* exactly analogous to σ_F^*; no η_E is mentioned because it would always be zero. Second, $\mathcal{A}_E$ starts equal to A_E, switches to A_E/S_R whenever $\dot{\phi}(t)$ changes from negative to positive, and switches back to A_E whenever $\dot{\phi}(t)$ changes from positive to negative.

The fourth level of analysis involves detailed specifications of how FASMOV actually calculates $\sigma_E(t)$ and $\sigma_E^*(t)$, and how FASMOV calculates or

selects an appropriate $\mathcal{H}(\cdot)$ function to use in each of its (3.4) and (3.5) integration operations. These specifications appear in Kilmer, et. al. (1981).

4. U-MODEL RESULTS FOR HINTR

Fig. 5 shows the excellent agreement between U-Model's computed response for Hintr1, and Hintr1's recorded responses shown in Fig. 2. U-Model's parameters were set to produce Fig. 5 as noted in the caption. The subject's data and U-Model's computed responses for the HintrA trial are presented in Figs. 6 to 7. The captions of Figs. 5 through 7 cover several salient points regarding the production of U-Model's outputs for Hintr1, and HintrA.

Since the U-Model is basically an integration model, it is not surprising that Figs. 2 and 6 differ much more between them than Figs. 5 and 7. If we define movement time as the time between the start of an arm movement and the first arrival at the 0° target position, Hintr's movement times are 140 ms in both Hintr1 and HintrA (such consistency is certainly not the rule!).

Fig. 8a) shows that the second flexor burst for HintrA is appreciably smaller than the first. But at the $\dot{\phi}$ sign change at 236 ms (cf. Fig. 7), the Q_F/Q_E ratio jumps to $(S_R)^2 = 9.0$ times its value immediately preceding 236 ms. The validity of the S_R idea here, as suggested by Heckathorne and Childress (1981), is strikingly borne out by the late stages of U-Model's response in Fig. 7. This excellent agreement between U-Model and subject data also suggests that the confounding effect of gravity (cf. Fig. 1), which was not included in our analysis, was insigificant.

Somewhat surprisingly, a constant electromechanical delay, μ, for both flexor and extensor gave best results in all our modelling. This is contrary to measurements taken between isolated experimental points by Lagasse (1975), for example, and underscores the difference between continuous dynamic and discrete initial parameter values. This difference is especially relevant since

relatively small changes in our model's timing parameters always led to quite large differences in the corresponding $\phi(t)$ output shapes.

5. THE J-MODEL FOR CHAN'S FAST TRIALS

Figs. 8 and 11 give the trial record and J-Model outputs for two of Chan's trials. J-Model is a slight augmentation of U-Model, as explained in the caption to Fig. 9.

As noted in Fig. 9, Chan's movements were very fast, and the dynamic operational characteristics of his muscles differed markedly from those of Hintr--so much so that the augmentation of our U-Model to the J-Model was necessary to enable the model to fit Chan's data at all well. The caption in Fig. 9 describes the augmentation.

From Figs. 9 and 11 we see that at T_{DE} a partial extensor desaturation occurs. Our interpretation of this is that the big extensor burst somehow partly "reprimes" the triceps.

At T_{DF} a more dramatic desaturation of the flexor occurs. This is accompanied by a sharp increase in the flexor tension-to-EMG scale factor, A_F, and a mild increase in the flexor saturation rate constant γ_F. The large increase in Reque (cf. caption, Fig. 9) means that the short twitch duration (i.e., small value of ξ_F) associated with the new σ_F^* at T_{DF} is maintained much longer than would be the case with the previous Reque of 0.3. Thus the second flexor burst produces a predominantly fast-twitch effect, but also an effect that saturates relatively quickly. This is discussed further below. Finally, note the small 38 value in T_{DF}'s summand; it implies that the priming effect of the second flexor burst became manifest in only about two thirds of an electromechanical coupling time.

6. DISCUSSION

Equations (3.1) to (3.5) give the basic structure of our model. In particular, (3.4) and (3.5) show that our major idea was to impose σ_F- and σ_E-controlled nonlinearities on $h_F(\cdot)$ and $h_E(\cdot)$, but otherwise to compute the Q_F and Q_E torques using the convolution integral of linear systems theory. Perhaps this notion has been entertained by others, but we mathematized it and put numbers into the equation. This gave us empirically inferred values for some skeletalmuscular functions that are valid for at least our movement. Heretofore such functions were determined only for essentially postural specifications or for small sinusoidal movements over linear ranges.

Our model assumes only feed-forward control (contrary to the ideas of Sakitt (1980) and Lestienne, et al. (1979)). This raises the question: given that Hintr's movement time was always 140 ms, how could the nervous system have known, e.g., that an A_E change from 0.28 on trial 10 of a block (Hintr1) to 0.32 on trial 14 of that block (Hintr2 of Kilmer et al. (1981)) was needed? The easiest answer is that the nervous system provided the same "drive energy" in both cases, but different muscle fibers received it: i.e., the spinal outflow and/or muscle inflow distributions changed with repeated movements, but the basic drive signals for the movement did not.

This answer is not necessarily applicable to the A_F-to-G_{AF} change at T_{DF} in Chan's movements. There we suspect that the nervous system fired more synchronously at T_{DF} than initially (cf. Milner-Brown, et al. (1975)) in addition to employing somewhat different motor units (or muscle fibers within motor units) at these times. The model's parameter changes at T_{DF} in Figs. 9 and 11 are what led us to this suspicion. Frequency analysis of raw EMG's should tell us more about this question soon.

In Chan trials 2, 4, 7, and 10, the movement times were 128 ms, 126 ms, 124 ms, and 120 ms respectively. This appears to be the result of some kind of "warmup" effect related to Chan's producing faster twitches throughout each trial than Hintr did. Note that Chan's initial A_F value changed from .28 in Chan2 to .21 in Chan10, whereas Hintr's initial A_F value changed in the opposite direction from .20 in Hintr1 to .23 in Hintr2. The foregoing relationships may enable us eventually to sharpen and better understand the correlations between muscle fiber types according to biopsy, muscle response speeds (or powers), and operant muscle twitch characteristics, at least for our type of movement. Kroll (1974) and others have sparse systematic evidence but abundant anecdotal evidence that the fast-twitch muscle fibers of biopsy are not always the fast-twitch ones of action. More work is in progress on this question.

Our model was quite sensitive to changes in parameter values, especially those that affected relative timing. The most disruptive changes of all were unbalanced flexor versus extensor ones. The antagonistic muscle systems in our bodies must have some way of balancing their changes due to temperature shifts, etc.; otherwise we could not retain the shapes of our movements when cold, hot, sick, or slightly inebriated.

We believe that our model performs essentially the same kind of integration that our elbow muscles do, at least when the EMG signals are clean and synchronous as in Hintr1 and A and Chan1 and 10. We also believe that the best-fitting parameter values arrived at in our Hintr and Chan runs are probably unique (to two decimals). Allowing for the coarseness of our model, which was retained to keep computer costs within reason, the fact that our model performed so well over many trials of several subjects gives it a credibility, especially in its temporal organization, approaching validation.

7. CONCLUSIONS

Our J-Model should prove useful in at least two ways. First, it should enable us to check further on the presently intrenched but increasingly suspect ideas on the role of biochemically classified fast-twitch and slow-twitch muscle fibers in fast movements such as ours. Some evidence to date suggests that the slow- and fast-twitch fibers of biopsy are not always the slow- and fast-twitch fibers of action. Now, by comparing J-Model simulation with muscle biopsy results, we can produce dynamically valid correlations on this question.

A second use of J-Model should be to aid the design of optimal electrical stimulation patterns for use in limb rehabilitation following strokes and other accidents. This would build on Boucher and Lagasse's (1980) success in transferring a skilled muscle activation pattern for our movement type from "donors" to unpracticed "recipients."

Fig. 1. Schematic representation of the essential elements involved in our arm movement. Initially ϕ is 60°, and the movement is to a target ϕ of 0°. The flexor is the biceps group, and the extensor is the triceps. Flexor torque is assumed equal to $\mathcal{T}_F A_F \cos\phi$, where $\mathcal{T}_F$ = flexor tension; and extensor torque is taken similarly as $\mathcal{T}_E A_E \cos\phi$.

Fig. 2. Plots from the dynograph recorder in Hintr's tenth trial on exercise day four: The top two traces are so-called "averaged EMG's," and are denoted $\widehat{\text{EMG}}(t)$ waves. d) biceps $\widehat{\text{EMG}}$; b) triceps $\widehat{\text{EMG}}$; c) ϕ displacement in degrees (cf. Fig. 1); d) $\dot{\phi}$ in degrees/sec; e) $\ddot{\phi}$ in degrees/sec^2. d) and e) are time-shifted to the right because of derivational delays. Millisecond time markers are written into the record at selected places, starting with 0 and the point where the a) plot rises off baseline. The small circles superposed onto the a) and b) plots are for deriving $\overline{\text{EMG}}$ inputs to the model from the dynograph $\widehat{\text{EMG}}$ plots. All Hintr1 runs were abbreviated to 340 ms to save computing costs. $\overline{\text{EMG}}_F(t)$ and $\overline{\text{EMG}}_E(t)$ represent the time courses of effective nervous drives on the flexor and extensor muscles respectively. The $\overline{\text{EMG}}$'s are essentially the same as the $\widehat{\text{EMG}}$'s except during the $\widehat{\text{EMG}}$'s falling phases. The continuous, piece-wise-linear waves formed by connecting adjacent pairs of encircled points in a) and b) are denoted $\overline{\text{EMG}}_F(t)$ and $\overline{\text{EMG}}_E(t)$ respectively. The $\overline{\text{EMG}}$'s are used to produce the $\overline{\text{EMG}}$'s as per Kilmer, et al. (1981). The idea is to let $\overline{\text{EMG}}$ equal $\overline{\text{EMG}}$ except where $\overline{\text{EMG}}$ has a negative slope, and then to let $\overline{\text{EMG}}$ be the level to which $\widehat{\text{EMG}}$ appears to be discharging through the dynograph's RC pen-control circuit.

Fig. 3. A pictorial outline of the conceptual basis of the U-Model. T_p is the time at which $\dot{\phi}$ switches from less than zero to greater than zero. S_R is the tension switching ratio for muscle lengthening vs. shortening. The S_R and $\mathring{n}_F$ values shown are fairly typical. In the Hintr1 run of FASMOV, our PASCAL implementation of U-Model, $S_R = 3.0$ and $\mathring{n}_F = -1.0$. The ":=" symbol means "is set to".

Fig. 4. a, b, and c show the successive $h_F(\cdot)$ functions used from the start of the Hintr1 run of U-Model until after a later reque of $h_F(\cdot)$ that sets it very close to its saturated state. d, e, and f show the same for $h_E(\cdot)$.

Fig. 5. The continuous curve is a replot of Fig. 2c) from curvilinear recorder coordinates to rectilinear coordinates. The small circles identify the U-Model's output as derived from its $\overline{EMG}_F(t)$ and $\overline{EMG}_E(t)$ inputs (cf. caption to Fig. 2). The parameters of U-Model were first set to best fit Hintr1's $\phi(t)$ response data. Then only A_E, A_F, and $\mathring{n}_F$ were allowed to be reset to best fit HintrA's data. That result is shown in Fig. 7.

Fig. 6. Similar to Fig. 2 except this Figure is for Hintr's fifteenth trial of the second block of trials on exercise day six.

Fig. 7. This Figure bears the same relation to Fig. 6 that Fig. 5 does to Fig. 2. Because HintrA's second flexor burst in Fig. 6a) was relatively large, we ran U-Model for 420 ms on HintrA.

Fig. 8. Similar to Fig. 2 except this Figure shows Chan's second trial on exercise day five. Chan's movement times for trials 2, 4, 8, and 10 of the same block that the above records are taken from were 128, 126, 124, and 120 ms (Hintr's were 140 ms on all trials). Chan was relatively very fast, and Hintr was about average.

Fig. 9. This Figure relates to Fig. 8 the same as Fig. 5 does to Fig. 2, except that it was produced by J-Model instead of U-Model. The two models differ as follows" let the first numbers in the T_{DE} and T_{DF} sums denote the onset times of the big triceps burst and the second big flexor burst respectively. The second numbers are empirical parameters. At T_{DE} we reset σ_E and at T_{DF} we reset five model parameters as shown at upper right in the Figure. Initial parameter settings are given at lower left in the Figure. The parameter values below the dashed line in the initial and reset parameter lists are the same for Chan2 and for Chan10 in Fig. 11; only the values above the dashed lines differ between Chan2 and Chan 10 runs. The value of Reque is the amount by which σ_F (or σ_E) must change since the last Reque operation (or since t=0) before another σ_F^* (or σ_E) reque is done. ξ_{CON} is discussed in Kilmer et al. (1981); it is used to reque ξ_F and ξ_E.

Fig. 10. Chan's trial 10 of the same block that the trial in Fig. 8 came from.

Fig. 11. This Figure relates to Fig. 10 the same as Fig. 9 does to Fig. 11. See caption for Fig. 9 for comments on the parameter lists shown in this Figure.

REFERENCES

Bizzi, E., Polit, A., Morasso, P.: Mechanisms underlying achievement of final head position. J. Neurophysiol. 39, 435-444 (1976)

Boucher, J.P., Lagasse, P.P.: Effect of functional electrical stimulation (FES) on neuromuscular coordination mechanisms. Research Report of the Laboratoire des sciences de l'activite physique, Universite Laval, Quebec, Canada (1980)

Cooke, R., Bialek, W.: Contraction of glycerinated muscle fibers as a function of the ATP concentration. Biophys. J. 28, 241-258, Nov. 1979

Desmedt, J.E., Godaux, E.: Voluntary motor commands in human ballistic movements. Ann. Neurol. 5, 415-421 (1979)

Dijkstra, Sj., Denier van der Gon, J.J.: An analog computer study of fast, isolated movements. Kybernetik 12, 102-110 (1973b)

Dijkstra, Sj., Denier van der Gon, J.J., Blange, T, Karemaker, J.M., Kramer, A.E.J.L.: A simplified sliding-filament muscle model for simulation purposes. Kybernetik 12, 94-101 (1973a)

Eisenberg, E., Hill, T.L., Chen, Yi-der: Cross-bridge model of muscle contraction: Biophysical Journal 29, 195-227 (1980)

Gydikov, A., Dimitrova, N., Kosarov, D., Dimitrov, G.: Influence of frequency and duration of firing on the shape of potentials from different types of motor units in human muscles. Exp. Neurol. 52, 345-355 (1976)

Hatze, H.: A general myocybernetic control model of skeletal muscle. Biol. Cybernetics 28, 143-157 (1978)

Hatze, H.: Neuromusculoskeletal control systems modelling--a critical survey of recent developments. IEEE Trans. on Auto. Control 25, 375-385 (1980)

Hatze, H.: Analysis of stretch responses of a myocybernetic model muscle fibre. Biol. Cybernetics 39, 165-170 (1980)

Heckathorne, C.W., Childress, D.S.: Relationships of the surface electromyogram to the force, length, velocity, and contraction rate of the cineplastic human biceps. Amer. J. Physical Med. 60:1, 1-19 (1981)

Houk, J.C.: Regulation of stiffness by skeletomotor reflexes. Annual Reviews of Physiol. 41, 99-114 (1979)

Kilmer, W., Kroll, W., and Congdon, V.: An EMG-level muscle model for a fast arm movement to target. ECE Dept. Report, UMass/Amherst, July(1981)

Kroll, W.: Fractionated reaction and reflex time before and after fatiguing isotonic exercise. Medicine and Science in Sports 6, 260-266 (1974)

Kroll, W., Clarkson, P.M.: Age, isometric knee extension strength, and fractionated resisted response time. Experimental Aging Research 4, 389-409 (1978)

Lagasse, P.P.: The prediction of maximum speed of human movement by two selected muscular coordination mechanisms and by maximum static strength and the effects of practice and fatigue upon maximum speed of human movement mechanisms. Unpublished Ph.D. dissertation, University of Massachusetts, Amherst (1975)

Lee, R.G., Tatton, W.G.: Motor responses to sudden limb displacement in primates with specific CNS lesions and in human patients with motor disorders. Canadian Journal of Neurological Sciences 2, 285-293 (1975)

Lestienne, F.: Effects of inertial load and velocity on the braking process of voluntary limb movement. Exper. Brain Res. 35, 407-418 (1979)

Lestienne, F., Polit, A., Bizzi, E.: From movement to posture. Psychology of Motor Behavior and Sport. Champaign, IL: Human Kinetics Publishers (1979)

Maton, B and Bouisset, S: The distribution of activity among the muscles of a single group during isometric contraction. European J. Appl. Physiol., 37, 101-109 (1977)

Messier, R. H., Duffy, J., Litchman, H.M., Paslay, P.R., Soechting, J.F., Stewart, P.A.: The electromyogram as a measure of tension in the human biceps and triceps muscles. Int. J. Mech. Sci. 13, 585-598 (1971)

Milner-Brown, H.S., Stein, R.B. Lee, R.G.: Synchronization of human motor units: possible roles of exercise and supraspinal reflexes. EEG and Clin. Neurophys. 38, 245-254 (1975)

Morris, Alfred F., Beaudet, Suzanne M.: Electromyographic latencies associated with rapid maximal force production in five different muscle groups in college adults. Amer. Corr. Ther. J., 34,-4, 116-119 (1980)

Norman, R.W., Komi, P.V.: Electromechanical delay in skeletal muscle under normal movement conditions. Acta Physiol. Scand. 106, 241-248 (1070)

Perrine, J.J., Edgerton, V.R.: Muscle force-velocity and power-velocity relationships under isokinetic loading. Med. Sci. Sports 10-3, 159-166, (1978)

Polit, A., Bizzi, E.: Characteristics of motor programs underlying arm movements in monkeys. J. Neurophys. 42, 183-194 (1979)

Sakitt, B.: A Spring Model and equivalent neural network for arm posture control. Biol. Cybernetics 37, 227-234 (1980)

Stein, R.B., Oguztoreli, M.N.: The role of -motoneurons in mammalian reflex systems. Biol. Cybernetics 39, 171-179 (1981)

Thorstensson, A., Grimby, G., Karlsson, J.: Force-velocity relations and fiber composition in human knee extensor muscles. J. Appl. Physiol. 40:1, 12-16 (1976)

Wadman, W.J., Denier van der Gon, J.J., Geuze, R.H., Mol, C.R.: Control of fast goal-directed arm movements. J. Human Movement Studies 5, 3-17 (1979)

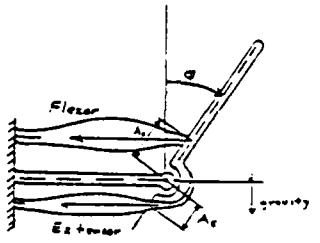
Wadman, W.J., Denier van der Gon, J.J., Geuze, R.H., Derksen, R.: Muscle activation patterns for fast goal-directed arm movements. J. Human Movement Studies 6, 19-37 (1980a)

Wadman, W.J., Boerhout, W., Denier van der Gon, J.J.: Responses of the arm movement control system to force impulses. J. Human Movement Studies 6, 280-302 (1980b)

Ward, Terry: Muscle state: reaction and movement time in elbow extension. Arch. Phys. Med. Rehabil, 59, 377-378 (1978)

Zuniga, E.N., and Simons, D.G.: Nonlinear-relationship between averaged electromyogram potential and muscle tension in normal subjects. Arch. Phys. Med., 50, 613-620 (1969)

1


Flexor
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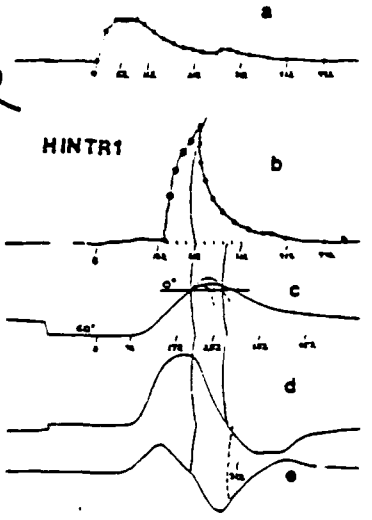
2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HINTR1
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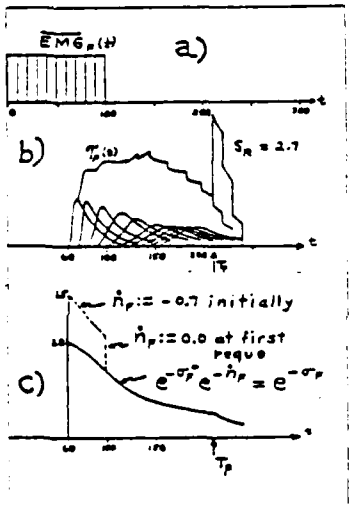
e

3


a)
b)
c)
$S_R = 2.7$
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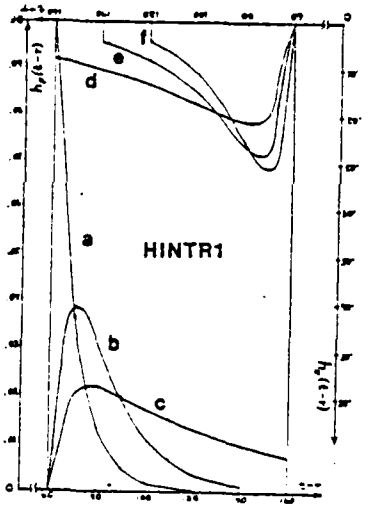
$\dot{n}_p := 0.0$ at first reque
$e^{-\sigma_p^*} e^{-\dot{n}_p} = e^{-\sigma_p}$

4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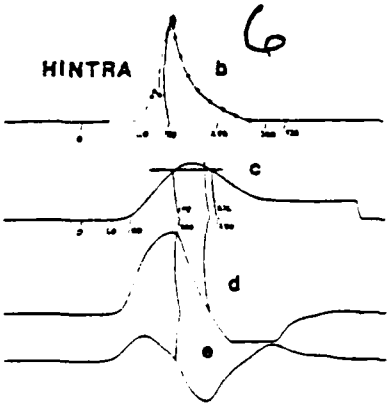
a

6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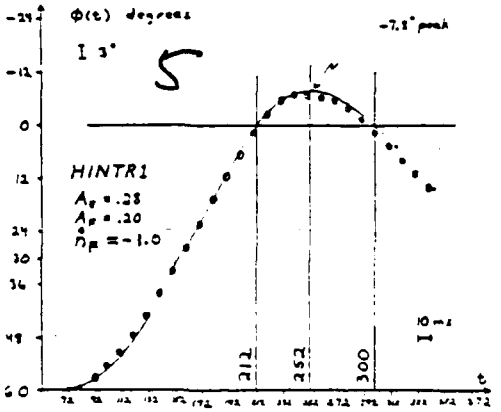
5


Φ(t) degrees
I 3°
HINTR1
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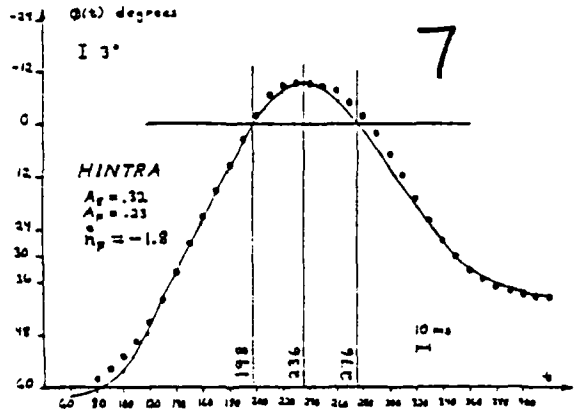
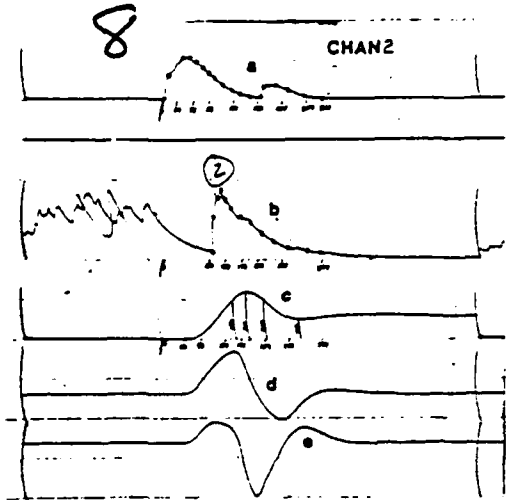
$\dot{n}_R = -1.0$
212
252
300
10 ms





$T_{DE} = 126 + 74 = 200$

$\sigma_E := (0.5)\,\sigma_E$

$T_{DP} = 242 + 38 = 280$

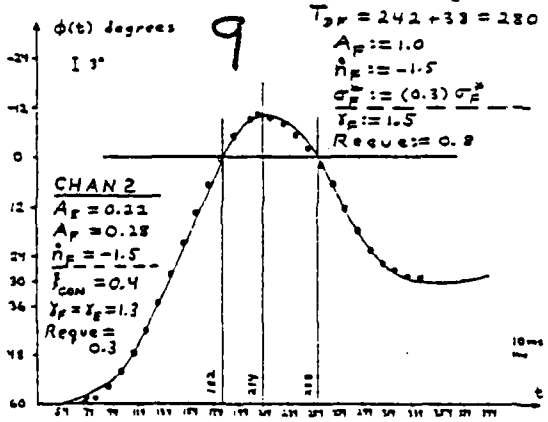
$A_P := 1.0$

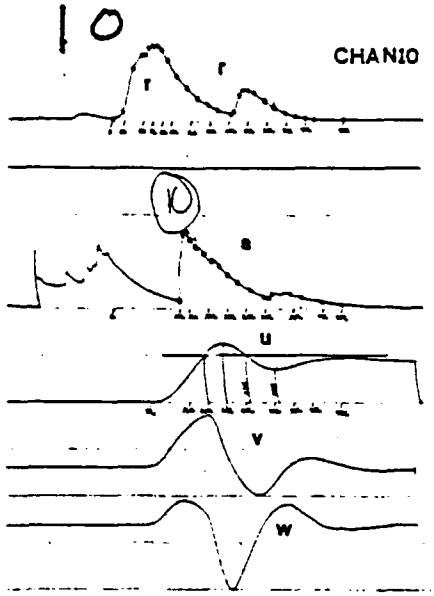
$\hat{n}_E := -1.5$

$\sigma_E^* := (0.3)\,\sigma_E^*$

$\gamma_E := 1.5$

$Reque := 0.8$





$T_{DE} = 146 + 70 = 216$

$\sigma_E := (0.6)\,\sigma_E$

$T_{DP} = 258 + 38 = 296$

$A_P := 0.4$

$\hat{n}_P := -0.7$

$\sigma_E^* := (0.5)\,\sigma_E^*$

$\gamma_P := 1.5$

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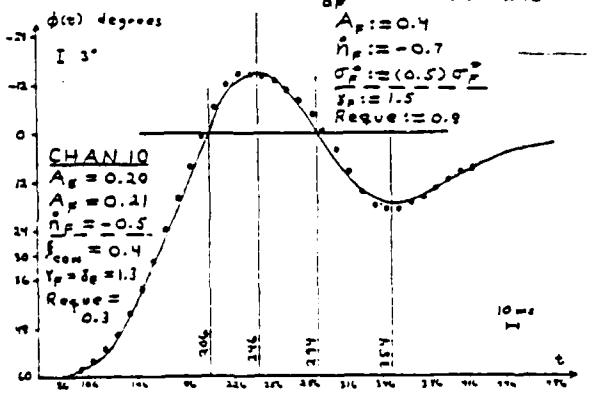

APPENDIX B

Article: On the Stability of Delay Equation Models of Simple Human Stretch Reflexes

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ON THE STABILITY OF DELAY EQUATION MODELS OF SIMPLE HUMAN STRETCH REFLEXES[#]

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ON THE STABILITY OF DELAY EQUATION MODELS OF SIMPLE HUMAN STRETCH REFLEXES

ABSTRACT

Human stretch reflexes (SRs) are often too weak and ineffectual to provide adequate postural regulation or rhythmic movement boosting (e.g. in ankle pushoff at the end of stance phase in fast running). Recent improvements in methods of artificially enhancing skeletomotor responses, especially in therapeutic regimens, should not be widely employed until the clonus-resisting stability properties of SRs are better understood.

We formulate an idealized linear servo model of a segmentally-mediated SR system which includes the often ignored electromechanical coupling delay. For typical closed-loop (delay/gain) ratios, the model is shown to be unstable for all values of loop gain when operating as a position servo, but maximally stable when operating as a velocity servo. We claim that the velocity servo or one of its nonlinear relatives is a better model for some well studied SRs than, e.g., Houk's stiff muscle hypothesis. We also present evidence that even feeble and quickly saturating monosynaptic postural servos are always unstable if operated as pure position regulators.

1. Introduction

Rack's (1981) critique of several papers on reflexes mediated by muscle afferents begins as follows:

> "The stretch reflex has often been regarded as part of a system for the servo-control of limb position, through which a discrepancy between the actual muscle length and an intended length generates activity that minimizes this error. If it is to work effectively, a servo-control system of this type must have a high gain around the feedback loop, so that a small error leads to a powerful correction; all the available measurements and calculations of this loop gain do, however, suggest that it is rather disappointingly low (Matthews, 1972; Vallbo, 1974) and this apparent shortcoming has sometimes puzzled physiologists...We are left therefore in a rather unsatisfactory position; there is ample anatomical and physiological evidence that the stretch reflex pathways exist, but a quantitative examination seems to indicate that they cannot do the job that we have frequently attributed to them."

In the same volume Hagbarth (1981), commenting on gamma loop reflex functions in man, says that it is

> "questionable whether the autogenetic reflex support via the (stretch reflex) loop ever exceeds what is required to counterbalance the force of gravity. With available data concerning the position sensitivity of spindles and their firing rate during isometric contractions, it has been estimated that the spindle inflow cannot be the main generator of the motor response that enables the subject to hold the joint position constant against increased external load."

Similar appraisals of the mammalian stretch reflex (SR) in its many guises abound in several professional journals. One wonders if SRs must be weak to be stable--i.e., to not respond clonically. With the ongoing development of ever more effective techniques for enhancing the power of SRs, especially in rehabilitative human limb therapy (cf. Kroll, et.al., (1981)), a direct mathematical study of the stability properties of spinal-segmentally mediated SRs (SSRs) as a function of their basic

elements, namely: feedback, gains, delays, and muscle mechanics, seems apropos. Though Evarts and Fromm (1981) argue convincingly that SRs in intact primates must always combine segmental and transcortical contributions to the changes in motorneuronal output following limb displacements, we still have SSR loops whose elements are artificially alterable, so our study seems justified as a precursor to a corresponding multiple-loop study (now in progress).

It is well known that by sufficiently increasing gain-delay products around simple closed loops, one can always eventually destabilize them. But whether the many weak SSRs that have been observed are close to instability has not been systematically studied. We shall formulate and analyze some simple (but we believe adequate) SSR models as evidence that, when typical relative parameter values obtain, the following principles hold: 1) If SSRs operate as pure velocity servos, they are always unstable. 2) If SSRs operate as pure velocity servos, they are stable provided the loop gain does not exceed a critical value. Velocity servos could act powerfully enough to fit the experimental data if their loop gains decreased fast enough after the SSR response began. 3) If SSRs operate in equal proportions as the sum of position and velocity servos, they have stability properties approximately midway between those of their two parts.

We shall also show that shortening loop delays greatly stabilizes the velocity servo. Finally, we present strong evidence that even mildly sensitive spindle afferents whose firing rates saturate quickly yield unstable postural controllers if the spindle activity is proportional to corresponding EMG levels.

2. Models to Date

To date only two series of papers provide mathematical frameworks sufficient to accommodate reasonably definitive investigations of the SSR stability-versus-power relationship. Hatze's series is formulated in terms of state equations for numbers of motor units in different contractile states, and contains too much fine-grained detail to be appropriate for our purpose (cf, e.g., Hatze (1980)). Stein and Oguztoreli's (1981) paper caps a series that analyzes loop responses to central nervous system inputs; but they assume an electromechanical coupling delay of zero and do their stability analysis as a function of delays in the spinal, cortical, and trans-cerebellar pathways (assumed to be 25ms, 55ms, and 85ms respectively). (Cf. Oguztoreli and Stein (1976) for the full stability analysis.)

We consider SSRs that contain an electromechanical coupling delay of perhaps 50ms and a spinal loop delay (from muscle receptor to corresponding EMG input to the receptor-bearing muscle) of, say, 25ms. We get different results than Stein and Oguztoreli, mostly because of our different delay structure but also because of different parameter values and muscle response shape. Delays such as we assume appear, for example, in Dietz et. al.'s (1979) study of gastrocnemius responses to foot contacts of sprinting humans. The stance phase in sprinting lasts only about 120ms, but the powerful terminating pushoff is apparently the result of an active tension in the gastrocnemius muscle caused by an SSR. For other examples, we assume that Houk's (1979) and Houk et. al.'s (1981) analysis of muscle stiffness applies to the short latency response

in Allum's (1981) study of load compensation in human ankle muscles under imposed foot rotations; so this and similar human SSRs fall within the same general time frame. Some of these are weak, and some appear to be moderately strong, at least if voluntary background activation levels are high and reflex durations are brief (Gottlieb and Agarwal, 1979).

Of particular interest to us because of our own experiments on falling (cf. Kroll, et.al. (1981)) is Dietz, et.al.'s (1981) study of triceps SRs in humans landing from forward falls. They suggest that the first high force peak just after contact with the landing platform is produced by a high initial muscle stiffness of Houk et.al.'s (1981) type, and that from about 70ms until about 200ms or so later, an SR fully activates the triceps to stop the fall. The first triceps EMG burst occurs at 20-30ms, signalling an SSR; a second EMG peak occurs at 60-80ms after touchdown, and this together with later peaks completes the SR. The elbow angle sags from about 150^{o} initially to about 90^{o} finally, without significantly increasing again until a later volitional response occurs.

If the oft-invoked concept of muscle acting as stiff spring is used here to explain Dietz et.al.'s triceps response, why weren't there significant elbow oscillations at the bottom of the fall? Surely the damping was not great enough to eliminate them. And why was the SR response not stronger? Unpublished results by Kroll show that electrical stimulation to the triceps just prior to such falls can decrease elbow bending angles and times to fractions of their normal values. Perhaps

Dietz et.al.'s SRs were not stronger because to have them so naturally would cause the triceps to often cramp off unstably. Could Dietz et.al.'s tricep SRs be better regarded as mediated by elbow angular-velocity servos of both SSR and long-loop types which zeroed elbow angular velocities as soon as stably possible after hand contact? This idea is compatible with the highly dynamic characteristics of most human SR systems. Or, could elbow-position servos that too feebly aimed to restore elbow angles at touchdown better explain Dietz, et.al.'s data?

Any attempt to confirm the validity of the above servo ideas, and then use it to artificially enhance SSRs for whatever purpose, should proceed cautiously. Houk, et.al. (1981) shows that small stretches give high spindle sensitivity, hence high loop gain. Gottlieb and Agarwal (1979) and Gurfinkel and Shik (1972) note that voluntary background activity level increases can multiply SSR loop gains severalfold. Marsden (1976) found that increasing the load on a finger muscle increased the associated SR. Vallbo (1981) suggests that this might have been caused by parallel activation of skeletomotor and fusimotor systems; but regardless of the mechanisms, the net effect was to increase SR loop gain. Proske and Walmsley's (1981) study of triceps surae stiffness in cats running freely on a treadmill shows that at treadmill speeds below 3 m/s the medial gastrocnemius (MG) acts somewhat like a spring in yoke with the soleus. But at higher speeds, MG acts much more powerfully, so its loop gain is then correspondingly higher. Data in Dietz, et.al. (1979) and in Dietz (1981) suggest that the same effect occurs in man. Gurfinkel and Shik (1972) and Wiesendanger (1978) point out the complex

tradeoffs between SSRs and long-loop SRs, especially supraspinally altered SRs. There are also abundant references on the distinction between ballistic reflexes and slow-tracking or fine-postural SRs. Some believe that SSRs are meant to function only as fine-postural position servos. Evarts and Fromm (1981) opine that "segmental and transcortical proprioceptive reflexes allow for servo controls which are important in generating 'mild' motor adjustments necessary for precise active movement and precise postural stability, but exteroceptive (rather than proprioceptive) signals trigger the intense muscular reactions which are often called for in response to stimuli arising from the environment."

With these caveats in mind, we shall formulate an SSR model that will enable us to investigate some SSR servo properties.

3. The General SSR Model

We consider an SSR system for extensor and flexor muscle groups about a single joint. Let F and E denote flexor and extensor, y the joint angle, $dy/dt = \dot{y} = x$ the joint's angular velocity, $dx/dt = \dot{x}$ the joint's angular acceleration, T_L the load torque about the joint (external torque plus damping), and T_F and T_E the torques due to flexor and extensor tension. Then, normalizing the moment of inertia for rotation about the joint to 1, we get

$$\begin{aligned} \dot{y}(t) &= x(t) \\ \dot{x}(t) &= T_F - T_E + T_L \,, \end{aligned} \tag{1}$$

assuming that extensor shortenings decrease y.

Kilmer et.al. (1981) have shown that at least for very fast, large amplitude, start-and-stop arm movements about the elbow, T_L and T_E can

be quite accurately expressed as a nonlinear convolution integral. We use here the linear approximation to this integral

$$T_E = \int_{t-\Delta_E}^{t} \overline{EMG}_E(\tau)\ h_E(t-\tau)d\tau, \qquad (2)$$

where $\overline{EMG}_E(\tau)$ is the envelope of the extensor EMG at time τ ; $h_E(t-\tau)$ is the extensor tension twitch at t corresponding to a unit impulse of $\overline{EMG}_E$ at $(t-\tau)$; and Δ_E is the duration of such a twitch.

A similar equation obtains for T_F. Focusing only on the negative feedback SSR, and supposing that moment arms, etc., remain constant throughout all rotations, we approximate (2) by

$$T_E = \int_{t-\Delta_E}^{t} \langle\langle b_E y(\tau - \delta) + c_E x(\tau - \delta) \rangle\rangle\ h_E(t-\tau)d\tau \qquad (3)$$

where δ is the time delay between an extensor stretch and the appearance on a corresponding extensor EMG, and $\langle\langle V \rangle\rangle$ is 0 if v is negative and is v otherwise.

The assumption in going from (2) to (3), namely that $\overline{EMG}_E(\tau)$ is a linear combination of $y(\tau - \delta)$ and $x(\tau - \delta)$, is discussed below, but for now we accept it on the supposition that y is at least approximately proportional to the difference between the present extensor length and a neutral reference length, and x likewise to the rate of change of length. These approximations seem tolerable in the light of our purpose, which is to characterize the nature of the relationship between SSR stability and loop gain and delay.

Our system is now linear, so if we set $h_F = h_E$, $b_F = b_E$, $c_F = c_E$, and $T = T_F - T_E$, we can rewrite (1) as

$$\dot{y}(t) = x(t)$$
$$\dot{x}(t) = -\int_{t-\Delta}^{t} \left[by(\tau - \delta) + cx(\tau - \delta)\right] h(t-\tau)\,d\tau \qquad (4)$$

We assume in (4) that T_L is 0 by dint of choosing appropriate reference levels and by taking damping to be negligible. We also assume in (4) that the quotient of muscle tension per unit $\overline{EMG}$ input in muscle shortening divided by that in lengthening is approximately the same for flexor as for extensor. The available evidence to date supports this assumption (cf. Kilmer, et.al. (1981)). Finally, we absorb δ into the h function in (4) and change variables to get

$$\dot{y}(t) = x(t)$$
$$\dot{x}(t) = -\int_{-\Delta}^{0} \left[by(t+\theta) + cx(t+\theta)\right] h(\theta)\,d\theta \qquad (5)$$

For analytical convenience we now rescale t to $\hat{t}$ so that $\hat{\Delta} = 1$, and approximate the shape and duration of $h(\theta)$ by

$$h(\theta) = \begin{cases} (1+\theta)\dot{h} & \text{for } -1 \leq \theta \leq T_h < 0 \\ 0 & \text{for } T_h < \theta \leq 0 \end{cases} \qquad (6)$$

where $\dot{h}$ is a positive constant. In most of what follows we let $T_h = 1/2$, with the interpretation that one time unit on the $\hat{t}$ scale is about 120ms on the t scale. The resulting $h(\theta)$ is interpreted to mean that a unit $\overline{EMG}$ impulse at t=0 leads to a twitch response which is 0 for about 50ms (the electromechanical coupling time), then jumps to $(1/2)\dot{h}$ at about 50ms, and then decreases linearly to 0 at about 120ms. This right triangular shape for a "twitch-like" response is a gross caricature of

real twitch shapes, but Kilmer, et.al. (1981) show that in the highly integrative muscle component of our system, such shape inaccuracies are not nearly as important as twitch starting time and area. $\dot{h}$ determines our twitch area, hence our loop gain. The final rendition of our SSR model is

$$\begin{aligned} \dot{y}(\hat{t}) &= x(\hat{t}) \\ \dot{x}(\hat{t}) &= -\dot{h}\int_{-1}^{-1/2} (\theta+1)\left[by(\hat{t}+\theta)+cx(\hat{t}+\theta)\right]d\theta \qquad (7) \\ &= f(by_{\hat{t}} + cx_{\hat{t}}) \end{aligned}$$

where the last line of (7) is the shorthand we use below. Henceforth, we shall write $\hat{t}$ simply as t.

4. A Velocity Servo

If b=0 and c=1 in (7), we have the velocity servo

$$\dot{x}(t) = -\dot{h}\int_{-1}^{-1/2} (\theta+1)x(t+\theta)d\theta = f(x_t) \qquad (8)$$

If we assume that the initial $x(\theta)$ data for $-1 \leq \theta \leq 0$ is piecewise continuous, it follows from Hale (1977) and the completeness properties of Fourier series that all solutions to (8) over any finite t interval are linear combinations of in general infinitely many terms of the form $\exp(\sigma_n t)$, where σ_n is the nth complex eigenvalue of (8): $\sigma_n = \gamma_n + i\beta_n$, n = 1,2... . Thus all solution terms are of the form

$$\exp(\gamma_n t)\left[A_n \cos\beta_n t + B_n \sin\beta_n t\right]$$

for appropriate constants A_n and B_n.

Suppose we order the σ_n so that

$$\gamma_1(\dot{h}) \geq \gamma_2(\dot{h}) \geq \gamma_3(\dot{h}) \geq \ldots$$

Referring to the solution term containing σ_n as the n^{th} mode, and that containing σ_1 as the dominant mode, we say (8) is stable if $\gamma_1(\dot{h}) \leq 0$, i.e., if the dominant mode is nonincreasing.

To find $\gamma_1(\dot{h})$ we substitute $\exp(\sigma t)$ into (8) for x(t) and solve the resulting eigenvalue equation, the real and imaginary parts of which are, respectively:

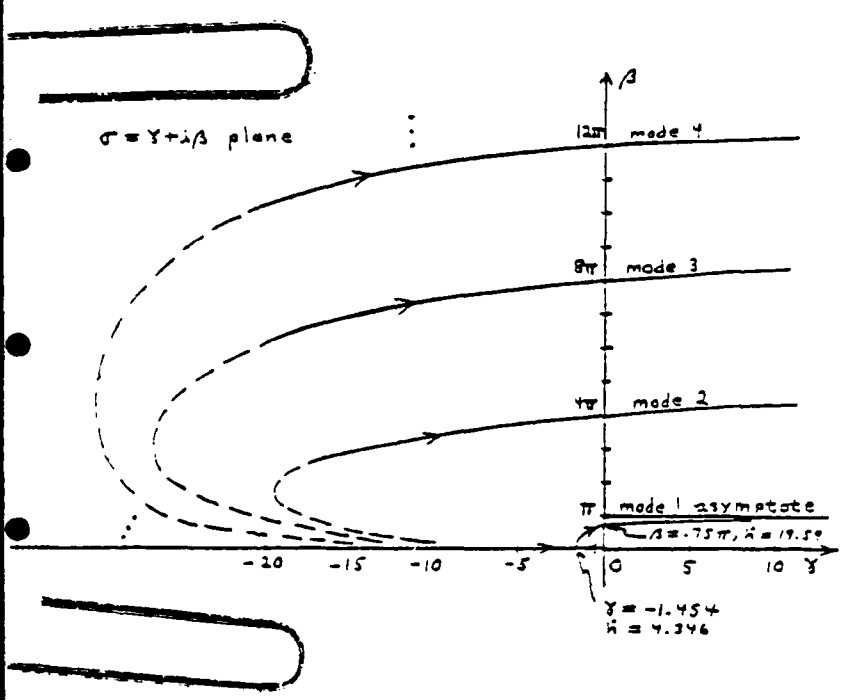
$$\left[\underline{(\gamma^2-\beta^2)}(\gamma^2+\beta^2)\exp(\gamma/2)\right]/\dot{h} =$$
$$-(1/2)(\gamma^2+\beta^2)\cos(\beta/2) + \gamma\cos(\beta/2) - \beta\sin(\beta/2) \qquad (9a)$$
$$-\left[\gamma\cos^2(\beta/2) - 2\beta\cos(\beta/2)\sin(\beta/2) - \gamma\sin^2(\beta/2)\right]/\exp(\gamma/2)$$

$$\left[\underline{2\gamma\beta}(\gamma^2+\beta^2)\exp(\gamma/2)\right]/\dot{h} =$$
$$(1/2)\sin(\beta/2) - \beta\cos(\beta/2) - \gamma\sin(\beta/2) \qquad (9b)$$
$$+\left[\beta\cos^2(\beta/2) + 2\gamma\cos(\beta/2)\sin(\beta/2) - \beta\sin^2(\beta/2)\right]/\exp(\gamma/2)$$

The underlined terms on the left side of (9a) and (9b) are referred to as $\underline{\Phi}_r$ and $\underline{\Phi}_i$ respectively below.

Figure 1 and Table 1 sketch the solutions to (9) as a function of $\dot{h}$. As noted in the Figure and Table captions, the two are slightly incompatible because of calculator roundoff errors. It is clear, however, that for an $\dot{h}$ of about 20 the dominant mode of the solution to (8) goes unstable. In order to translate this into response shapes for different $\dot{h}$ values in a hypothetical idealized falling situation, we computed the response shown in Figure 2 on a CDC Cyber using the highly accurate algorithm of Banks and Kappel (1979). For comparison, we also show there as x*(t) a sketch derived from Dietz et.al.'s (1981) data on forward falls. x*(t) is a crude approximation to the angular velocity at an elbow; t = -0.3 is touchdown time; and the triceps SR presumably provides most of the braking force to stop the falls (cf. Figure caption).

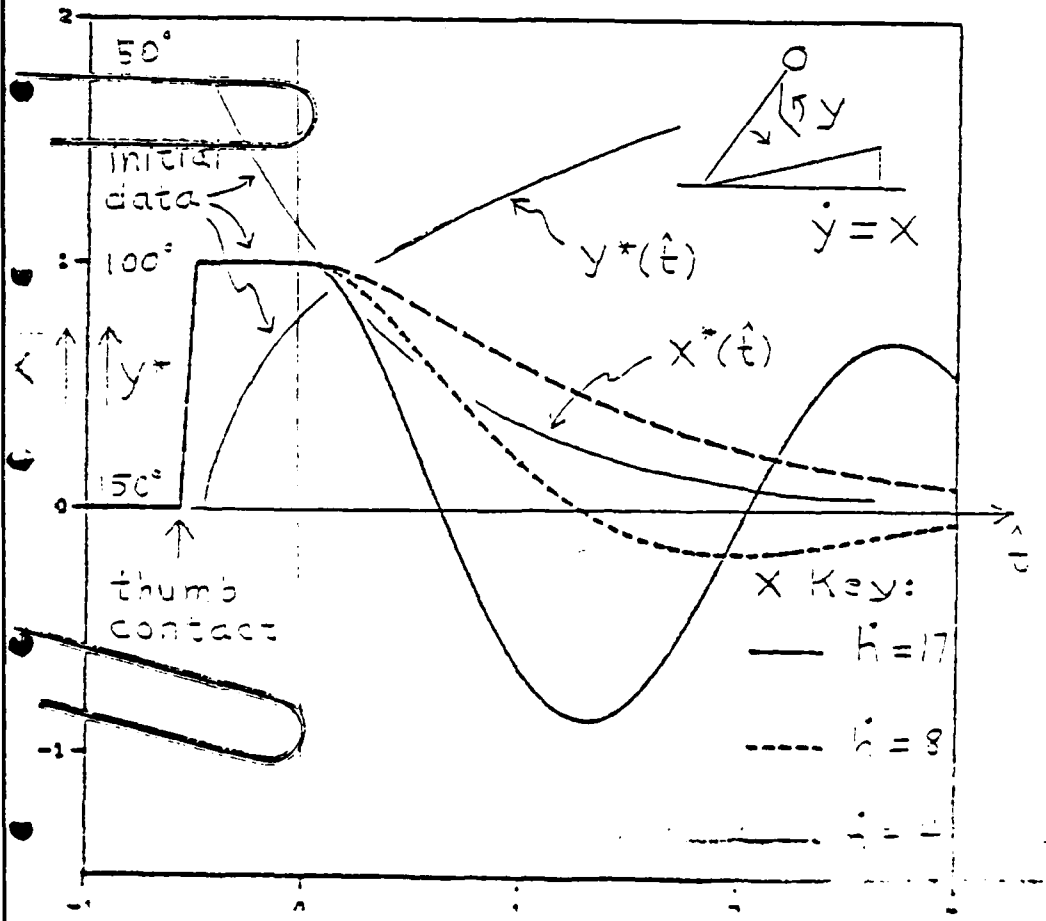
Figure 1. Sketch of the eigenvalue loci in the top half of the σ plane for the $x = \exp(\sigma t)$ solutions to $\dot{x} = f(x_t)$. The arrows on all loci point in the direction of increasing $\dot{h}$, as seen from Table 1. The scale on the dashed loci is distorted to keep the loci in the Figure. The bottom half of the σ plane is the mirror image of the top half.



$\dot{h}$	γ	β
MODE 1 (dominant mode)		
$\dot{h} \to 0^-$	$\gamma \to 0^-$	0
2.861	-0.500	0
4.078	-1.000	0
4.319	-1.300	0
4.346	-1.454	0
4.347	-1.4538	0.01π
4.452	-1.432	0.1π
5.062	-1.315	0.25π
8.15	-0.868	0.5π
18.69	-0.0495	0.74π
19.59	0	0.75π
20.43	0.0416	0.76π
31.93	0.507	0.85π
81.43	1.534	π
817.04	4.36	1.25π
4.5×10^{4}	10.06	1.50π
9.1×10^{3}	26.32	1.75π
2.0×10^{20}	74.80	1.9π
MODE 2		
0.0005	-16.95	2.40π
44.38	-2.75	3.50π
315.8	0	4π
MODE n, n > 2		
1.2×10^{3}	0	8π
.	0	$\left\{ 4\pi(n-1) \right\}_{n=4,5,\ldots}$

Table 1. $\dot{h}$ versus the $\sigma = \gamma + i\beta$ eigenvalue for the $x = \exp(\sigma t)$ solutions to $\dot{x} = f(x_t)$. All values were found by gradient search using an HP41C hand calculator. Roundoff errors are a few percent for $\dot{h} < 30$, but large for large $\dot{h}$. This accounts for the discrepancy between Table 1 and Figure 1. The first row symbols in the Table mean "$\dot{h}$ goes to 0 through values > 0", and "γ goes to 0 through values < 0".

Figure 2. Solutions to (8) for different $\dot{h}$ values as computed on a CDC Cyber using Banks and Kappel's (1979) algorithm. A 16-spline approximation was used. The initial data for $t < 0$ is interpreted as the angular velocity at an elbow joint after touchdown (at $t = -.5$) of a man falling as shown in the stick-figure insert. The hypothetical velocity servo of equation (8) does not develop any braking tension in the triceps until $t = 0$ (50ms after touchdown in the interpretation). $y^*(t)$ is a sketch, on a slightly different scale for readability, of data from Dietz et.al.'s (1981) Figure 2. $x^*(t)$ is our crudely derived approximation to $\dot{y}(t)$, and is reasonably accurate only for $t > 0$. Experiments by us indicate that our equation (8)'s initial data is a better idealization of what one usually finds.



Note the comparison to our $x^*(t)$ sketch of the corresponding Dietz et.al. (1981) data. Obviously, by letting $\dot{h}$ in (8) decrease appropriately after $t = 0$, we could fit the $x^*(t)$ curve exactly. Any reasonable interpretation of $x^*(t)$, though, would have to suppose that the initial falloff of x^* (from touchdown at $t = 060$ms (or $t = -0.5$) until the vicinity of 0 was due to a passive viscoelastic effect, and that only the subsequent falloff of x^* could have been controlled by our hypothetical velocity servo. Given the strong, long-lasting pre-innervation of the triceps before touchdown (especially in the blindfolded subjects), a decreasing h after $t = 0$ due to a saturation effect seems plausible as a way to represent the decreasing (tension/$\overline{EMG}$) ratios that must inevitably occur in such fast, high-powered arm movements (cf. Kilmer, et.al. (1981)). That such an h saturation would be stabilizing to about the right degree can be seen by comparing the shape of the x^* response in Figure 2 with the other responses there. We believe that this is the best way to conceive of Dietz et.al.'s findings, not only for the SSR response component, but for the entire SR response. We warn, therefore, of the attendant danger of instability if artificial stimulation is employed. Appendix A proposes an analytically tractable way of letting h decrease so as to yield a response like Dietz et.al.'s.

To see how important the relative timing in our equation (8) model is, we show Figure 3. Shortening the loop delay is a powerful way of stabilizing the system; this should be kept in mind whenever considering SSRs that occur in muscles that are already strongly preinnervated (hence quickened in their electromechanical coupling). Appendix A shows,

interalia, that in the extreme case where Δ in Figure 3 is 0, the system is asymptotically stable, with no solution overshoots (i.e. sign changes) even if the initial data is either all positive or all negative.

Figure 3 suggests why Stein and Oguztoreli's (1981) assumption of a shortest loop delay of only 25ms could yield a falsely secure stability analysis. Note, however, that Oguztoreli (1976) graphically portrays how his long-loop 55ms and 85ms delays can be stabilizing if oscillatory signals over his long-loop paths arrive at the muscle in antiphase with those over his short-delay path. Nowhere do Stein or Oguztoreli consider pure velocity servos.

5. A Position Servo

If $b = 1$ and $c = 0$ in (7), we have the position servo

$$\dot{y}(t) = x(t) \tag{10a}$$

$$\dot{x}(t) = -h \int_{-1}^{-1/2} (\theta + 1) y(t+\theta)\, d\theta = f(y_t) \tag{10b}$$

Proceeding as in the previous Section, we obtain eigenvalue equations that differ from (9a) and (9b) in that for (10) we have

$$\bar{\Phi}_r = \gamma(\gamma^2 - 3\beta^2) \tag{11a}$$

$$\bar{\Phi}_i = \beta(3\gamma^2 - \beta^2) \tag{11b}$$

Figure 4 and Table 2 sketch the solution to the eigenvalue equations as a function of $\dot{h}$. The roundoff errors are somewhat larger than with the velocity servo, but the qualitative nature of the solution space for equation (10) is that indicated in Figure 4. The central result is that our position servo is unstable for all positive values of $\dot{h}$. Figure 5

Figure 3. Solutions of $\dot{x} = f_{\Delta}(x)$ for three different Δ s to show the stabilizing effect of reducing the delay Δ. $\dot{h} = 20$ for all curves. The $\Delta = 1/2$ curve (not shown), has an envelope whose magnitude increases very slowly. These curves were obtained using a hand calculator.

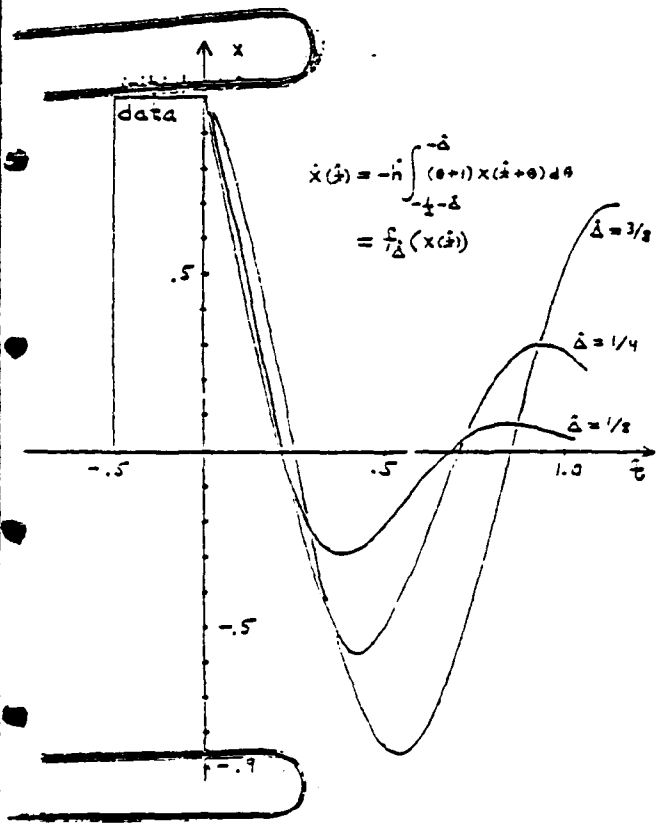
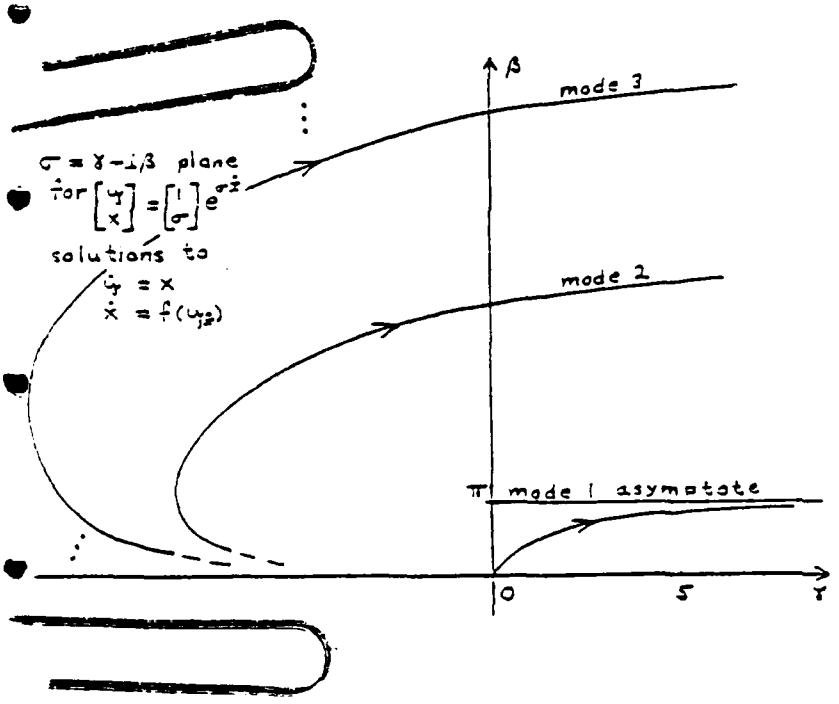


Figure 4. Similar to Figure 1 only now $\dot{x} = f(y_t)$. Again there is a countable infinity of modes. Modes higher than the first are not drawn to scale.



$\dot{h}$	γ	β
	MODE 1	
0	0	0
0.996	0.040	0.11π
6.10	0.210	0.25π
14.73	0.418	0.35π
21.96	0.552	0.40π
47.53	0.887	0.50π
103.35	1.321	0.60π
360.0	2.20	0.75π
911.0	2.90	0.85π
4,512.	4.51	π
5.3×10^{7}	16.81	1.5π
	MODE 2	
0.2966	-11.19	1.5π
12.67	-5.86	2π

Table 2. Similar to Table 1 except now $\dot{x} = f(\gamma_t)$.

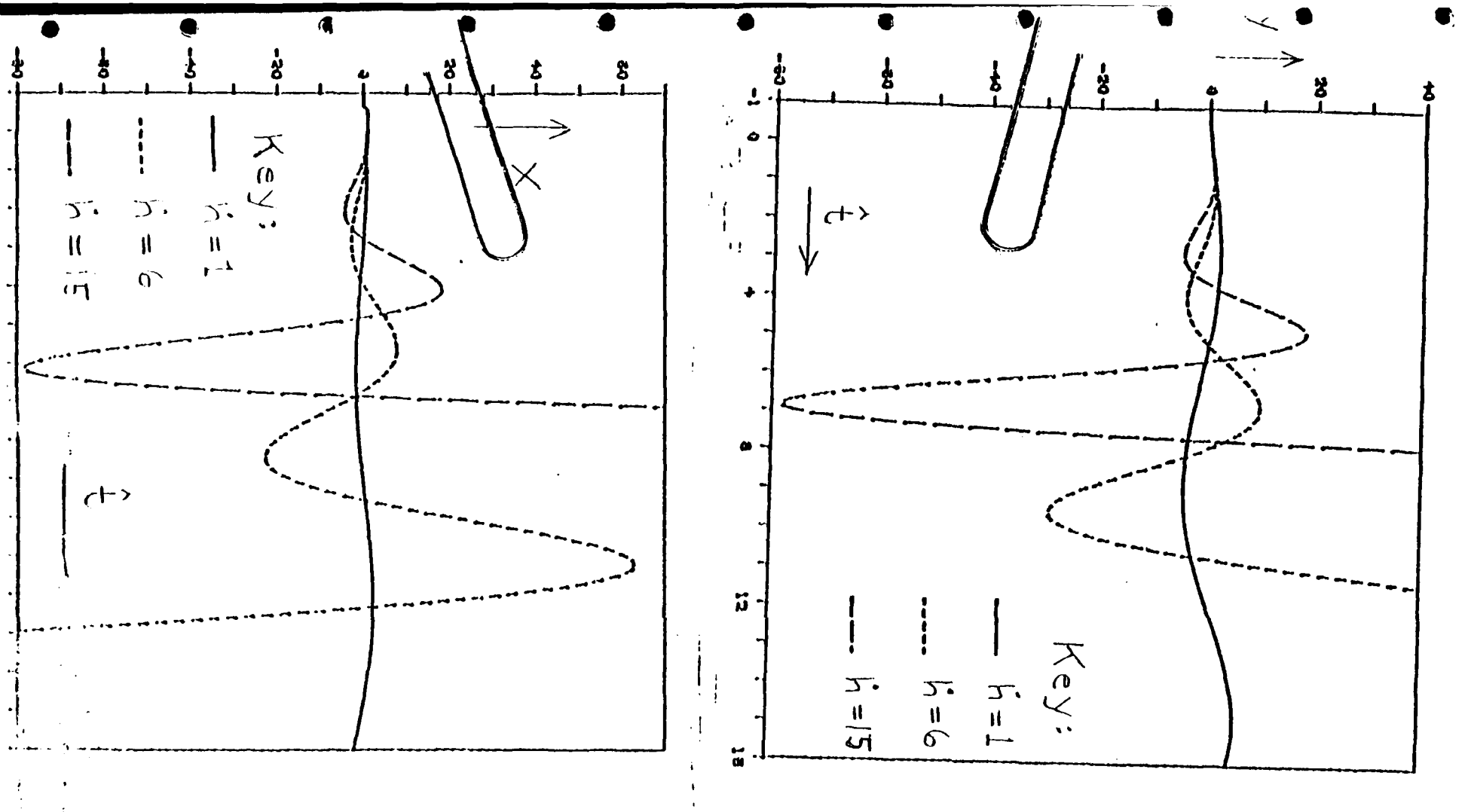


Fig. 5. Similar to the x curves in Fig. 2 except now $\dot{x} = f(y_t)$. The same initial data was used here as for the x curves in Fig. 2, with $y(-.5) = 0$ and $y(0) = .5$.

shows some solutions of (10) for different $\dot{h}$ values to convey this more graphically. Thus pure linear position servos should never be sought in the body's melange of SSRs. This should eliminate the "length follow-up servo" hypothesis of Merton (1953) because that postulated a fusimotor equivalent of stretch as the actuator of a response otherwise produced by a system essentially the same as (10).

6. A Combined Position and Velocity Servo

If b = 1 and c = 1 in (7), we have

$$\dot{y}(t) = x(t) \tag{12a}$$

$$\dot{x}(t) = -\dot{h} \int_{-1}^{-1/2} (\theta+1)\left[y(t+\theta) + x(t+\theta)\right] d\theta = f(y_t + x_t) \tag{12b}$$

The eigenvalue equations for (12) differ from (9) in that for (12) we have

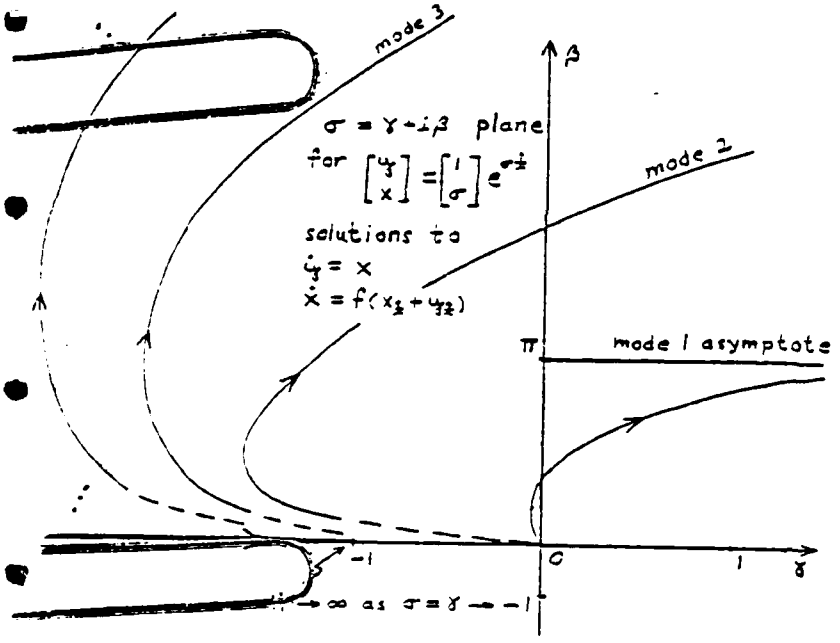
$$\overline{\omega}_r = \frac{\gamma(\gamma^2-3\beta^2)(\gamma+1) + \beta^2(3\gamma^2-\beta^2)}{(\gamma+1)^2+\beta^2} \tag{13a}$$

$$\overline{\omega}_i = \frac{\beta(3\gamma^2-\beta^2)(\gamma+1) - \beta\gamma(\gamma^2-3\beta^2)}{(\gamma+1)^2+\beta^2} \tag{13b}$$

Figure 6 and Table 3 briefly sketch the eigenspace for (12) as a function of $\dot{h}$. The roundoff errors are larger than ever, but the qualitative features in Figure 6 are correct. Figure 7 plots solutions to (12) for several different $\dot{h}$ values. Our combined servo gives us an expected blend of its component servo response characteristics. Differently proportioned b and c values would simply weight the two component characteristics differently to produce the total system response.

The combined servo is less stable than the velocity servo, but in its stable range of gains it takes the position error asymptotically to

Figure 6. Similar to Figure 4 except now $\dot{x} = f(x_t+y_t)$. Again there is a countable infinity of modes. Modes higher than the first are not drawn to scale.



$\dot{h}$	γ	β
	MODE 1	
3.0	-0.0426	0.213π
7.365	-0.023	0.364π
20.0	0.468	0.627π
50.0	1.31	0.842π
250	3.05	π^-

Table 3. Similar to Table 2 except now $\dot{x} = f(x_t + y_t)$.

zero, albeit too slowly and with too much oscillation to believe any SSRs actually operate that way. See the graphs in Appendix B.

Shortening the delay in (12) from 1/2 to $\Delta < (1/2)$ has much the same effect as it did for (8). In fact, if $\dot{h}$ is also greatly decreased (to slow the servo down), we can use the results of Driver (1976) to show that (12) behaves increasingly like

$$\dot{y}(t) = x(t) \qquad (14)$$
$$\dot{x}(t) = (-h/2)(y(t) + x(t))$$

whose eigenvalues are $-(\dot{h}/4) \pm (\dot{h}/4)(1-4/\dot{h}/2)^{\frac{1}{2}}$. Thus for $(\dot{h}/2)^{\frac{1}{2}} < 4$, (14) gives stable oscillations, where otherwise its solutions are of the form $c_1 \exp(-\alpha_1 t) + c_2 \exp(-\alpha_2 t)$, with $\alpha_1, \alpha_2 > 0$ and c_1, c_2 appropriate constants. It can be shown in general that reducing the product of gain and delay in (12) always has a stabilizing effect.

Quite possibly SSRs of the type (12), but approaching the sluggish limit (14), operate in respiratory, masticatory, or other systems where responses can be slow and positions are not always corrected by volitional acts.

7. Discussion

Houk et.al. (1981b) found that in decerebrate cats the discharge rate r of spindle receptors to large ramp increases in length obeys asymptotically the relationship

$$r - r_o = K(y-y_1)x^{0.3} \qquad (15)$$

where K and y_1 are constants and r_o is the discharge rate before stretching begins. If (15) were proportional to our $\overline{EMG}$ in (2) and x and y were always positive, (7) would change to

$$\dot{y}(t) = x(t)$$
$$\dot{x}(t) = -\dot{h} \int_{-1}^{-1/2} K\left[y(t+\theta)-y_1\right] \left| x(t+\theta) \right|^{0.3} (\theta+1)\, d\theta \qquad (16)$$

Note that (16) assumes linear (spindle activity)-to-EMG and EMG-to-muscle tension relations.

Matthews (1981) has questioned the generalizability of (15) to other than ramp stretches, but concedes that $(r - r_o)$ always increases in response to a stretch far more slowly than in direct linear proportion to the stretch velocity. This is contrary to (7), but Houk et.al. (1981) suggest that the end result of (14) may be a force-length relation around the SSR loop which is nearly linear over a restricted physiological range, in line with his stiff-spring hypothesis. In fact, Gottlieb and Agarwal (1979) found a linear relationship between velocity of imposed stretch and magnitude of reflex EMG into human triceps surae, but the brief 10-40ms duration of their myotatic reflex was referred to by Houk et.al. (1981b) as a linearly scaled initial discharge at the beginning of the latters' several-second ramp stretch.

We believe that (7) may be an acceptable compromise for at least a restricted range of physiological conditions, but we also believe that (16) merits further study. Noting that in Houk's reply to Matthews' comments above, Houk said (15) does not hold during muscle shortening, we set K in (16) to K_+ when $\dot{x} \geq 0$ and K_- when $\dot{x} < 0$, with $K_- = 5K_+$ (the "5" here was an educated guess). The numerical solution of the resulting equation, starting from the initial data of Fig. 2, is shown in Fig. 7 for two different $\dot{h}$ values. Evidently, (16) behaves essentially

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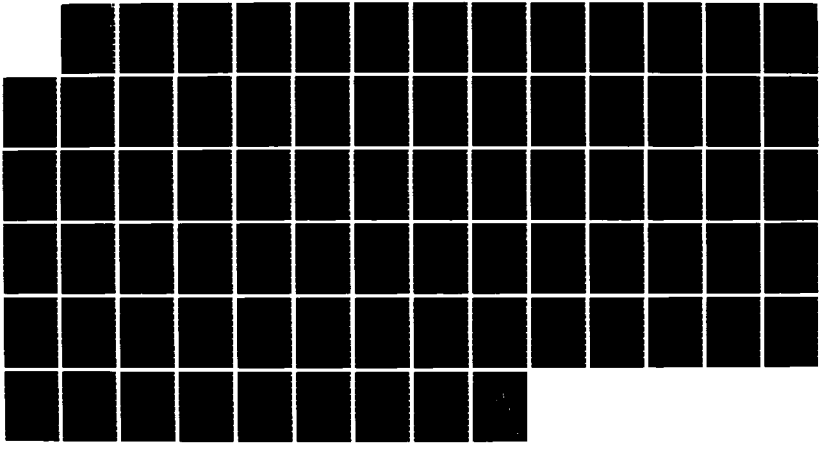


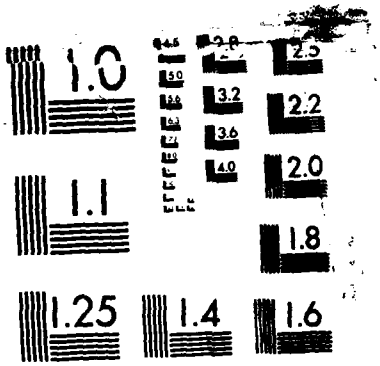
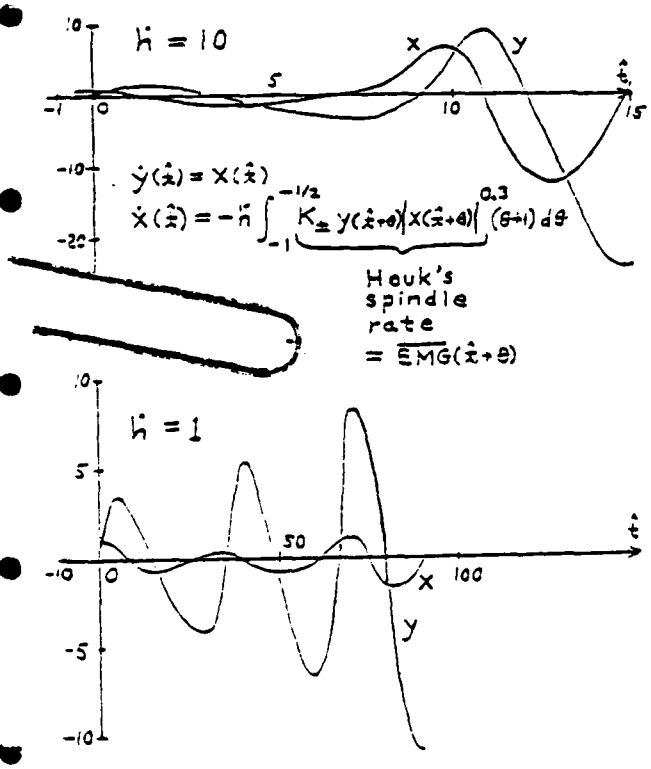


Figure 7. Numerical solution to the equation shown, as calculated with a hand calculator using 16 steps per t time unit and a Simpson's integration rule for the right side of the $\dot{x}(t)$ equation. Note the different time scales in the $\dot{h} = 10$ and $\dot{h} = 1$ plots.



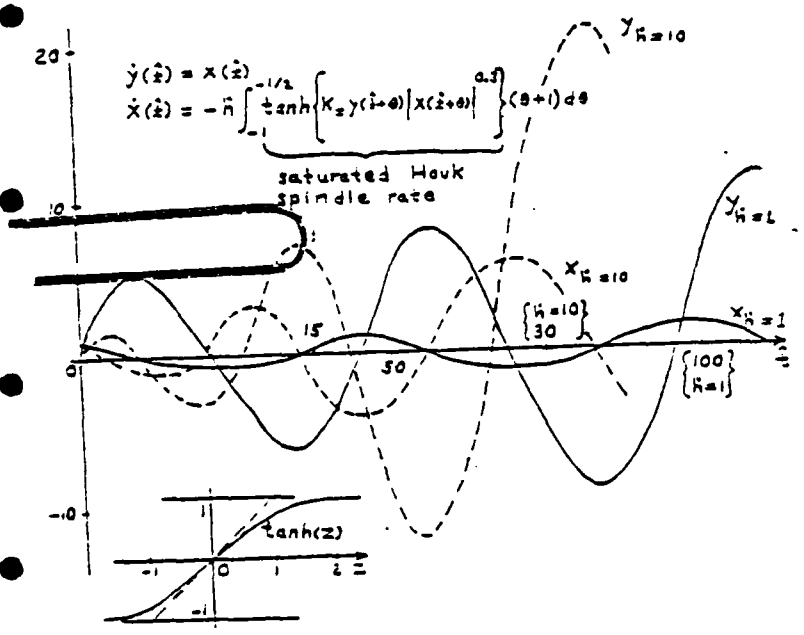
like a position servo, (the small exponent on $|x(t+\theta)|^{0.3}$ keeps this factor close to 1). Thus (16) is probably unstable for all positive values of $\dot{h}$.

Since spindle firing rates reach their upper limit quite easily, we augmented (16) with the tanh saturating function shown at lower left in Fig. 3, and again solved the resulting equation numerically. As Fig. 8 shows, this did not change the general character of the solution at all. Thus even mildly sensitive and quickly saturating spindle drives on α-motorneurons are very unstable in the position-like servo of Fig. 8. This fact is in stark contrast to what one often reads in the physiological literature.

A future paper will treat the extension of (7) to include not only the SSR loop but also the two long loops of Oguztoreli (1976). We shall analyze the resulting system as various combinations of velocity and position servos operating with various combinations of loop gains and as perturbed by stochastically shifting delays. Oguztoreli and Stein have not considered such combinations and stochasticity in their series of papers.

A natural test of (7) would be to apply it to the active component of the gastrocnemius push-off tension in sprinting humans. The challenge would be to factor the stance-phase triceps surae SR into passive viso-elastic effects, possible $\dot{h}$ boosting effects at the beginning of each step's gastrocnemius SSR response (especially likely in fast-stretch muscular athletics), fatigue effects, and the possible effects of properly timed electrical stimulation to improve performance.

Figure 8. Similar to Fig. 7 only the $\dot{x}(t)$ equation now contains the tanh saturating function shown at lower left. The $\dot{h} = 10$ scale appears above the t axis and the $\dot{h} = 1$ scale appears below it.



8. Conclusion

Our linearized SSR model, equation (7), is unstable as a position servo, and stable at sufficiently small gains as a velocity servo. We propose that the velocity servo formulation fits Dietz's triceps SR data for falling humans better than Houk's stiff-muscle hypothesis. Some other SRs, such as occur in biceps when an arm in handshake position is suddenly loaded, should also eventually model best as velocity servos because probably they are among the more powerful clonus-free SR systems of the type described by (7).

A position-like SR servo suggested by Houk's power law for spindle firing rates was shown to be unstable even for mild spindle sensitivities and quickly saturating firing rates, contradicting some of the physiological literature (but none of Houk's papers).

Presently we have strong analytical and simulation evidence that the pure position servo of (10) with one or two long loops added is unstable for all positive gain and delay sets, and that the corresponding velocity servos are stable as long as the sum of the gain-delay products for all loops is less than a fairly tight bound. This suggests the possibility that all control of postural steadiness is mediated by the following: steady, centrally produced signals; velocity or mixed velocity and position servos (accounting for the ubiquitous 10 Hz. tremor); and volitional "saccades".

Our results should apply to the design of functional electrical stimulation regimens, and to the possible future design of muscle-power amplifiers.

REFERENCES

Allum, J. H. J.: Observations on the control of human ankle position by stretch flexes. In: Muscle Receptors and Movement, Taylor, A., Prochazka, A. (eds.). London, Basingstoke: MacMillan Publishers Ltd. (1981).

Banks, H. T.: Approximation of nonlinear functional differential equation control systems. Journal of Optimization Theory and Applications, Vol. 29, No. 3, pp. 383-418 (November 1979).

Banks, H. T., Kappel, F.: Spline approximation for functional differential equations. Journal of Differential Equations, Vol. 34, No. 3, pp. 496-522 (December 1979).

Dietz, V.: Contribution of spinal stretch reflexes to the activity of leg muscles in running. In: Muscle Receptors and Movement, Taylor, A., Prochazka, A. (eds.). London, Basingstoke: MacMillan Publishers Ltd. (1981).

Dietz, V., Noth, J., Schmidtbleicher, D.: Interaction between pre-activity and stretch reflex in human triceps brachii during landing from forward falls. J. Physiol., 311, 113-125 (1981).

Dietz, V., Schmidtbleicher, D., Noth, J.: Neuronal mechanisms of human locomotion. Journal of Neurophysiology, Vol. 42, No. 5, pp. 1212-1221 (September 1979).

Driver, R. D.: Linear differential systems with small delays. Journal of Differential Equations, Vol. 21, No. 1, pp. 148-166 (1976).

Evarts, E. V., Fromm, C.: Transcortical reflexes and servo control of movement. Canadian Journal of Physiology and Pharmacology, Vol. 59, No. 7, pp. 757-775, (July 1981).

Gurfinkel, V. S., Shik, M. L.: The control of posture and locomotion. In: Motor Control, pp. 217-234, Gydikov, A. A., Tankov, N. T., Kosarov, D. S. (eds.). New York: Plenum (1973).

Hagbarth, K. E.: Fusimotor and stretch reflex functions studied in recordings from muscle spindle afferents in man. In: Muscle Receptors and Movement, Taylor, A., Prochazka, A. (eds.). London, Basingstoke: MacMillan Publishers Ltd. (1981).

Hale, J.: Theory of functional differential equations. Berlin, Heidelberg, New York: Springer-Verlag (1977).

Hatze, H.: Neuromusculoskeletal control systems modeling--A critical survey of recent developments. Transactions on Automatic Control, Vol. AC-25, No. 3, pp. 375-384 (June 1980).

Houk, J. C., Crago, P. E., Rymer, W. Z.: Function of the spindle dynamic response in stiffness regulation - a predictive mechanism provided by non-linear feedback. In: Muscle Receptors and Movement, Taylor, A., Prochazka, A. (eds). London, Basingstoke: MacMillan Publishers Ltd. (1981).

Houk, J. C.: Regulations of stiffness by skeletomotor reflexes. Ann. Rev. Physiol., 41, 99-114 (1979).

Houk, J. C., Rymer, W. Z., Crago, P. E.: Nature of the dynamic response and its relation to the high sensitivity of muscle spindles to small changes in length. In: Muscle Receptors and Movement, Taylor, A., Prochazka, A. (eds.). London, Basingstoke: MacMillan Publishers Ltd (1981).

Kilmer, W., Kroll, W., Congdon, V.: An EMG-level muscle model for a fast arm movement to target. Exercise Science Department Report, University of Massachusetts/Amherst (1981).

Kroll, W., Lagasse, P., Kilmer, W.: Functional electrical stimulation, sensory imparted learning and neuromotor coordination mechanisms: Relevance to therapeutic limb rehabilitation regimens. Internal Report, Exercise Science Department, University of Massachusetts/Amherst (1981).

Marsden, C. D., Merton, P. A., Morton, H. B.: Servo-action in the human thumb. J. Physiol., London 257, 1-44 (1976).

Matthews, P. B. C.: Critique. In: Muscle Receptors and Movement, Taylor, A., Prochazka, A. (eds.). London, Basingstoke: MacMillan Publishers Ltd (1981).

Merton, P. A.: Speculations on the servo-control of movement. In: The Spinal Cord, Wolstenholme, G. E. W. (ed.). London, Churchill, 247-255 (1953).

Oguztoreli, M. N., Stein, R. B.: The effects of multiple reflex pathways on the oscillations in neuro-muscular systems. Journal of Mathematical Biology 3, 87-101 (1976).

Rack, P. M.: Critiques. In: Muscle Receptors and Movement, Taylor, A., Prochazka, A. (eds.). London, Basingstoke: MacMillan Publishers Ltd (1981).

Stein, R. B., Oguztoreli, M. N.: The role of γ-Motoneurons in mammalian reflex systems. Biol. Cybern. 39, 171-179 (1981).

Taylor, A., Prochazka, A. (eds.): Muscle receptors and movement. Proceedings of a Symposium held at the Sherrington School of Physiology, St. Thomas' Hospital Medical School. London, Basingstoke: MacMillan Publishers Ltd. (1981).

Wiesendanger, M.: Comments on the problem of transcortical reflexes. J. Physiol., 74, 325-330 (1978).

Appendix A

A LIAPUNOV STABILITY ANALYSIS FOR FIGURE 3 ALSO GIVES ONE FOR AN x*-APPROXIMATING SYSTEM FOR FIGURE 2.

The title of this Appendix states its purpose.

The system in Figure 3 is

$$\dot{x}(t) = -h \int_{-1/2-\Delta}^{-\Delta} (\theta + 1) x(t+\theta)\, d\theta \tag{A1}$$

We call such a system stable if for every piecewise continuous initial data $x(\theta)$ such that the maximum $|x(\theta)|$ for θ in the interval from $-1/2-\Delta$ to 0 inclusive is δ, there exists an $\epsilon(\delta)$ such that the maximum $|x(t)|$ over all finite $t \geq 0$ is $\leq \epsilon(\delta)$.

In Section 5.3, Hale (1977) considers the equation

$$\dot{x}(t) = -\int_{-r}^{0} a(-\theta)\, g(x(t+\theta))\, d\theta \tag{A2}$$

where all of

$$a(r) = 0,\ a(t) \geq 0,\ \dot{a}(t) \leq 0,\ \ddot{a}(t) \leq 0,\ 0 \leq t \leq r \tag{A3}$$

are continuous, and

$$G(x) = \int_{0}^{x} g(s)\, ds \to 0 \text{ as } |x| \to \infty \tag{A4}$$

He shows that the function

$$V(x(t)) = G(x(t)) - 1/2 \int_{-r}^{0} \dot{a}(-\theta) \left[\int_{\theta}^{0} g(x(t+s))\, ds \right]^2 d\theta \tag{A5}$$

is Liapunov; that is, that

$$dV(x(t))/dt = 1/2\ \dot{a}(r)\left[\int_{-r}^{0} g(x(t+\theta))d\theta\right]^2$$
$$-1/2\int_{-r}^{0} \ddot{a}(-\theta)\left[\int_{\theta}^{0} g(x(t+s))ds\right]^2 d\theta \qquad (A6)$$

is $\leqslant 0$ for all finite $t \geqslant 0$ (our assumption of piecewise continuous initial data is an obvious generalization of his continuous initial data). Thus by his Corollary 3.1, (A2) is stable.

If $\Delta = 0$, equation (A1) is of the form (A2)-(A4); hence it is stable. Moreover, if its initial data is either always positive or always negative, its solution is easily shown to have always the same sign as its initial data.

When $\Delta > 0$, as it must be in our SSR interpretation, Hale's functional does not yield (A6). Applying (A5) to (A1) with $\Delta = 1/2$, $r = 1$, $a(-\theta) = (\theta+1)\dot{h}$, $\dot{a}(-\theta) = \dot{h}$, $\ddot{a}(-\theta) = 0$ and $g(x) = x$, we get

$$dV(x(t))/dt = -1/2\ \dot{h}(-1)\left[\int_{t-1}^{t-1/2} x(u)du\right]^2 \qquad (A7)$$
$$+ \dot{x}(t)\left[x(t)-x(t-1/2)\right]$$

Routine manipulations show that $dV(x(t))/dt \leqslant 0$, implying that (A1) is stable, if

$$x(t-1)/x(t) \leqslant 8\ \dot{h} \text{ for all } t \geqslant 0 \qquad (A8)$$

(a loose sufficiency condition).

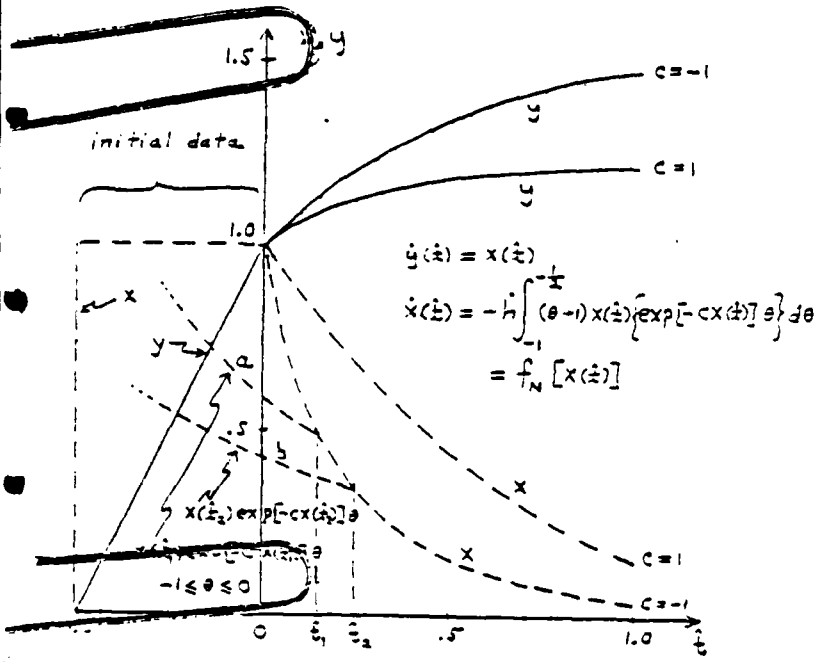
Assuming a solution to (A1) of the form $\exp(-\alpha t)$, with $(-\alpha)$ the dominant negative real eigenvalue of (A1), (A8) implies $\exp(\alpha) \leqslant 8\ \dot{h}$, where $\dot{h}$ here cannot exceed the largest value permitted by a positive real in (A1)'s eigenvalue equation

$$\alpha^2 = \dot{h}\exp(\alpha/2)\left[(\exp(\alpha/2)-1)/\alpha - (1/2)\right] \qquad (A9)$$

This $\dot{h}$ is 4.346, as seen from Figure 1, and the associated α satisfying (A9) is 1.454. Since $\exp(3.549) = 8(4.346)$, this associated α easily satisfies (A8). A simple argument also shows that for initial data of the form in Figure 3, $dV(x(t))/dt$ in (A7) is < 0 for all $t > 0$. Thus we have a Liapunov characterization of solutions to (A1) that go asymptotically to 0 as t get large. This characterization will now be used to prove the stability of a nonlinear modification of (A1).

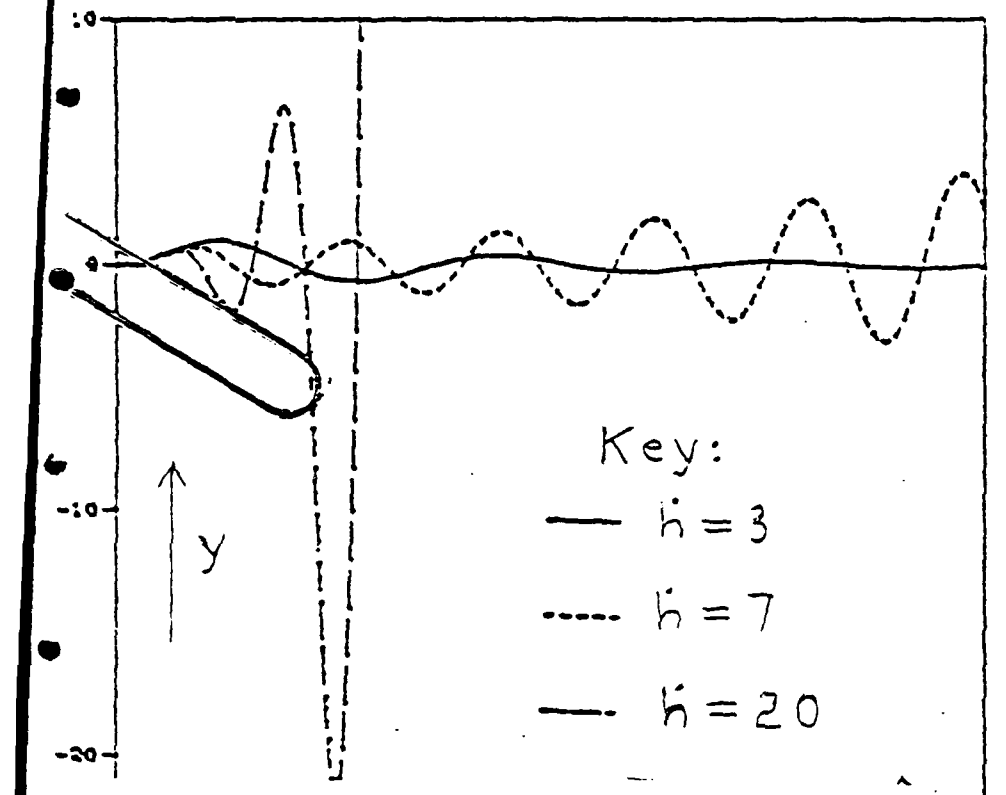
The modification is given in Figure A1 as $f_N(x(t))$. The idea of f_N is to have a form of the type (A1) but such that $\dot{h}$ is now an original constant $\dot{h}$ times $\exp(cx(t))$ for some real constant c. This would give a stable solution to $\dot{x} = f_N(x)$ by the above Liapunov argument if at each $x(t)$ the solution's backward continuation over $-1 \leq \theta \leq 0$ were $\exp(cx(t))$ instead of its larger true value. Replacing $x(t+\theta)$ in (A1) by the $x(t)\left[\exp(-cx(t))\theta\right] = x(t)\left[\exp(-\alpha_t \theta)\right]$ factor in $f_N(x(t))$ accomplishes precisely this change. The results for two values of c are plotted in Figure A1 for ease in visualization.

Figure A1. Solutions to $\dot{x} = f_N(x)$ as determined by hand calculator with Δt step size equal to 0.05. The backward curves a and b from $x(t_1)$ and $x(t_2)$ with c = -1 portray graphically how the integrand in $f_N(x)$ decreases with decreasing x.



Appendix B

Response Plots for Equation (12)



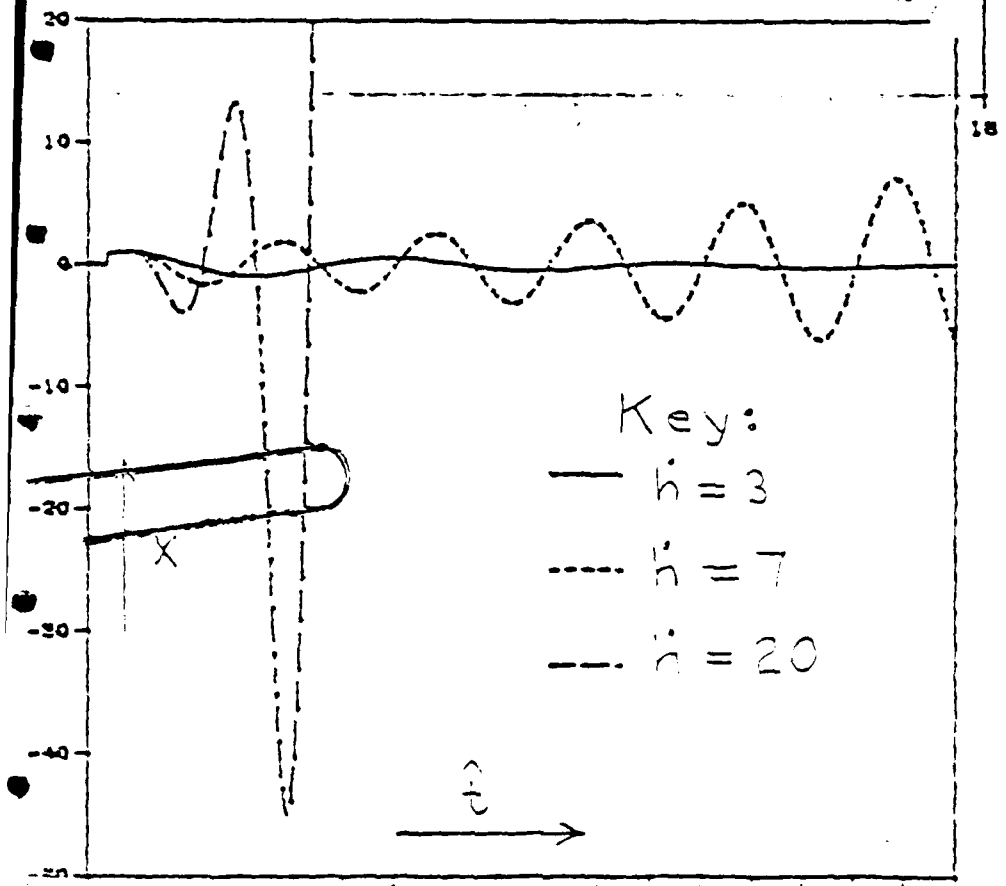


Fig. B1. Similar to Fig. 5, only now $\dot{x} = f(x_t + y_t)$. Same initial data as in Fig. 5.

APPENDIX C

Article: Functional Electrical Stimulation, Sensory Imparted Learning, and Neuromotor Coordination Mechanisms: Relevance to Therapeutic Limb Rehabilitation Programs

Presented at Neuropnysiology Symposium on Movement Disorders, Columbia University, March 12-16, 1982.

APPENDIX C

ARTICLE

FUNCTIONAL ELECTRICAL STIMULATION, SENSORY IMPARTED LEARNING AND NEUROMOTOR COORDINATION MECHANISMS: RELEVANCE TO THERAPEUTIC LIMB REHABILITATION PROGRAMS

Functional Electrical Stimulation, Sensory Imparted Learning, and Neuromotor Coordination Mechanisms: Relevance to Therapeutic Limb Rehabilitation Regimens

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Introduction

The terms muscle reeducation, gait retraining, and neurological training commonly used in rehabilitation exemplify the importance of motor skill development in therapeutic exercise regimens. In hemiplegic patients, for example, rehabilitation is considered to be a learning process involving reorganization of the central nervous system in order to relearn lost functions (Swenson, 1978). In cerebral palsy, there is a deficiency of motor control and the goal of rehabilitation is to help the patient acquire new motor skills (Halpern, 1978). It is clear, therefore, that the success of therapeutic exercise systems is closely linked to motor control and motor learning knowledge domains. That is, an understanding of the mechanisms of motor control and motor learning is crucial to the development and prescription of therapeutic exercise systems.

As new information becomes available in the neurophysiology of movement, researchers in motor control and motor learning attempt to assimilate this new information into existing theories and models. In some instances, the new information verifies and clarifies existing theory. In other instances, of course, the new information necessitates correction of existing theory. In still other instances, the new information may suggest the wisdom of abandoning current theory and the development of new theory.

Recent discovery of sensory imparted learning has led to the postulation of a new theory of motor learning, the reverse loop theory of motor learning. None of the existing motor learning theories make any provision for sensory imparted learning, suggesting the potential significance of the reverse loop theory for all aspects of motor learning. The reverse loop theory of

motor learning, furthermore, is not simply a modification of existing motor learning theories, but an entirely new type of motor learning. Coupled with new information about neuromotor coordination mechanisms of fast limb movements and functional electrical stimulation, it appears that a suitable theoretical interface can be proposed which has considerable relevance for therapeutic exercise systems.

It is the purpose of this paper to present a brief overview of the relevant new developments in motor control and learning, neuromotor coordination mechanisms, and functional electrical stimulation as they relate to therapeutic exercise systems.

Neurophysiologically Based Exercise Regimens

A number of central nervous system pathologies produce a variety of residual motor deficits which are aggravatingly resistant to rehabilitation. Because of less than satisfactory results with traditional exercise regimens, newer therapeutic exercise systems have been developed which are based largely upon neurophysiological mechanisms underlying volitional muscular activity. Analytical comparisons of the newer and more promising therapeutic exercise systems (Harris, 1978; Hirt, 1967; Flanagan, 1967) suggest that these systems exhibit marked similarities in terms of actual techniques employed, and even greater similarities in terms of central nervous system effects being sought. As seen in Table 1, each of the more recognized neurophysiologically based exercise systems is characterized by a subset of specific sensory stimulation techniques capable of facilitating or inhibiting volitional muscular action. Other systems utilize inherent movement patterns which are designed to simulate particular stages of normal motor development.

TABLE 1

THERAPEUTIC EXERCISE SYSTEMS BASED ON NEUROPHYSIOLOGICAL PRINCIPLES

THERAPEUTIC EXERCISE SYSTEM	MODALITIES	PURPORTED MECHANISM
Ayres (1973) and Frostig (1970)	Sensory stimulation	Re-integration of sensory-motor system
Bobath (1970)	Neurodevelopmental: postural reflexes, equilibrium reactions, stretch, tapping, reflex inhibiting posture	Movement potential released by inhibition of spasticity
Brunnstrom (1970)	Homolateral and bilateral associated reactions	Central facilitation, peripheral proprioception, cutaneous stimulation
Fay (1954)	Pathological and unlocking reflexes	Primitive movement patterns unlock brought under voluntary control
Frenkel ataxia coordination (1902)	Repetitive patterning	Education of CNS via sensory stimuli
Kabat (1965)	Maximal contractions, reflexes, irradiation, successive induction	Proprioceptive neuromuscular facilitation
Knott and Voss (1962)	Spiral and diagonal movement patterns with stretch and repetition	Proprioceptive neuromuscular facilitation
Phelps (Jones, 1967)	Massage, passive motion, active assistive motion, active motion, resisted motion, balance	Ontogenetic approach to mimic normal physical development stages
Rood (1954)	Cutaneous stimulation of discrete skin areas by brushing, icing, stretch	Proprioceptive facilitation
Zankel (1960)	Electrical stimulation assisting voluntary contraction	Neuromuscular reeducation

Although not explicitly stated, the basic principle common to all neurophysiologically based exercise systems is the goal of transmitting sensory stimulus patterns which are more intense than that produced without the added proprioceptive facilitation. Frenkel's coordination exercises for tabetic ataxia (1902), for example, emphasizes repetition as a means of producing desirable changes in the central nervous system:

> The treatment for tabetic ataxia is based upon the education of the central nervous system by means of repeated exercises, whereby it is enabled to receive sufficiently distant stimuli from the limbs...
>
> Theoretically, the transformation of an ataxic movement into a normal movement takes place in tabetic subjects according to the same laws as the acquisition in healthy persons of a complicated movement...

Thus, Frenkel's system emphasizes repetitive movement as a means of transmitting appropriate sensory stimulus patterns to the central nervous system with such repetitive movements chosen to simulate fundamental stages of normal motor development in an appropriate sequence. Quite similar rationales are espoused by treatment regimens prescribed by Fay (1954), Rood (1954), Phelps (1967), and Doman-Delacato (Delacato, 1966: Doman et al., 1960).

The Bobath system (1970) utilizes various postural reflexes and equilibrium reactions "...for purposes of modifying muscle tone or eliciting desired movements (Harris, 1978)." Muscle stretch and tapping are used to further enhance proprioceptive facilitation. Herman Kabat was one of the first proponents of proprioceptive neuromuscular facilitation as a "... valuable tool in therapeutic exercise to increase central excitation and thereby enhance the voluntary activity of paralyzed or paretic muscles

(Knott and Voss, 1962)." Included in Kabat's suggestions were techniques utilizing reflexes, irradiation, and successive induction.

It seems clear, therefore, that the production of enhanced sensory stimulus patterns for transmission to the central nervous system is the goal of all modern therapeutic exercise systems based upon neurophysiological mechanisms. Implicit in such a treatment rationale is the importance of transmitting appropriate sensory stimulus patterns of enhanced intensity. Beneficial effects of such heightened sensory stimulation transmitted to the central nervous system are presumed to enhance recovery. The exact neural mechanisms involved remain poorly defined but most theoretical explanations include some variation of the concept of neuronal plasticity (Bach-y-Rita, 1980; Cotman, 1978). The neuronal plasticity argument, of course, purports that the central nervous system has the capacity to create alternate pathways and thus compensate for damage to previously established central nervous system pathways for volitional muscular activity.

It was once held that damage to central nervous system pathways and centers was irreversible. Evidence has accumulated, however, which suggests that recovery of central nervous system functional capabilities may occur in several ways. Cotman, Nieto-Sampedro, and Harris (1981), for example, have shown that actual formation of synapses may result from repeated stimulation, and suggested that such "reactive synaptogenesis" may be a mechanism involved in recovery. Synaptic plasticity can also manifest itself by actual morphologic changes in synaptic structures as Eccles (1979) has shown an increase of up to 40 percent of synaptic spines on dendrites of granule cells in hippocampal neurons. More extensive dendritic trees

of layer II pyramidal neurons have been described in the elderly (Buell and Coleman, 1976) suggesting plasticity even in the aging brain. As early as 1902, Lazarus speculated that recovery of function in hemiplegia might result by compensatory utilization of different nerve pathways which take over for damaged pathways. Both Wall (1980) and Bach-y-Rita (1981) suggest that existing but unused pathways may be "unmasked" when the ordinarily used pathways have been damaged. Such unmasking is enhanced by an increased central excitability due to decreased sensory input. Proprioceptive facilitation is also known to allow the recruitment of "subliminal fringe neurons" which cannot be volitionally activated. It should be noted that the sensory stimulus pattern transmitted to the central nervous system plays an essential role in all of the proposed mechanisms for plasticity described above.

Equally obvious is the observation that a limb (or any movement synergy) affected by some pathological condition can produce only inefficient and incorrect volitional movement. Such inefficient and incorrect volitional movement produces a sensory stimulus pattern for transmission to the central nervous system which is itself inefficient and incorrect. Addition of proprioceptive stimulation techniques may improve the quality of the movement produced, but only to a small degree. Thus, even with the benefit of proprioceptive neuromuscular stimulation, the central nervous system still receives an impoverished sensory stimulus pattern. If recovery of central nervous system functioning is dependent upon the quality of the sensory stimulus pattern received, then recovery is likely to be slow and dependent upon gradual improvement in the quality of the movement synergy which produces slightly improved sensory stimulus patterns.

What, however, would be the effect upon central nervous system functioning if the sensory stimulus pattern transmitted to it from an afflicted limb was not only correct and efficient, but an optimized sensory stimulus pattern representing "perfect" limb movement patterns? Such a possibility now seems feasible because of developments in functional electrical stimulation, in the understanding of neuromotor coordination mechanisms for fast human limb movements, and in the demonstration of sensory imparted learning. As will be shown, each of these three relatively distinct areas of research have certain elements of relevance to each other, and a blending of such elements may hold promise for a vastly improved rehabilitation regimen for recovery of function in afflicted limbs.

Functional Electrical Stimulation

In 46 A.D. a Roman physician named Largus used the electrical discharge of torpedo fishes in the treatment of pain caused by headache and gout. A rival Greek physician, Dioscorides, discounted the value of torpedo fishes for headache relief but recommended its efficacy in the treatment of hemorrhoids. In 1745, the Leyden jar and an electrostatic generator were used to treat angina pectoris, epilepsy, hemiplegia, kidney stones, and sciatica. Benjamin Franklin used an electrical device to successfully treat a young woman suffering from convulsive fits. Electrical stimulation has also been used in hydroelectric baths to cure chronic inflammation of the uterus, and, in the 1920s, used in conjunction with a spiritualist to drive out a possessing spirit from the body of a mentally disturbed patient.

Fortunately, such an amusing early history of the simple beginnings of electrical stimulation did not prevent eventual development of a wide

variety of useful therapeutic and rehabilitative techniques. Cardiac pacemakers and transcutaneous electrical nerve stimulation (TENS) for the treatment of pain, for example, are two such valuable developments. Another electrical stimulation technique receiving major attention is that of functional electrical stimulation (FES). Work in FES involves stimulation of the nervous system or the muscle in order to restore lost function. Liberson, for example, developed both the technique and the device allowing stimulation of the peroneal nerve to activate the anterior tibialis muscle and help restore a normal gait pattern in hemiplegic patients with a foot drop problem.

At present, two major centers of research and development are active in the field of FES. Under the leadership of Lojze Vodovnik, the Faculty of Electrical Engineering at the University of Ljublana in Yugoslavia have concentrated their efforts in the engineering development of electrical stimulators to be used as prosthetic devices. The Rehabilitation Engineering Center, Rancho Los Amigos Hospital, University of Southern California has a similar focus with added emphasis upon treatment and the education of practitioners in the use of FES techniques. FES is currently being used in the treatment of patients with stroke, arthritis, spinal cord injury, and post-operative orthopedic problems.

FES and Neurological Recovery

Limb paralysis is included among the numerous ailments for which electrical stimulation has been prescribed ever since Johann Gottlog Krueger speculated about its use for restoration of function in paralyzed limbs in 1743 (Licht, 1959). As described by McNeal (1977), Kratzenstein in 1744,

Jallabert in 1748, and Quelmalz in 1753 all reported successive restoration of function in paralyzed limbs by the use of electricity. The modern era of the use of electrical stimulation seems to have begun with classical study reported by Liberson and his colleagues in 1961[1]. In 1961, Liberson and associates successfully developed a technique which stimulated the peroneal nerve when the heel of the foot of a hemiplegic patient touched the floor. Stimulation of the peroneal nerve activated the anterior tibialis muscle and helped restore a normal gait pattern to hemiplegic patients with a foot drop condition. It was observed that even without benefit of the electrical stimulation "...the patients acquire the ability of dorsiflexing the foot by themselves." Although Liberson and his associates noted that the positive effects were transitory, the concept of a "functional electrotherapy" in which electrical stimulation of a muscle could replace impaired innervation was born.

Other investigators (Carnstam, Larsson, & Prevec, 1977; Dimitrijevic, Gracanin, Prevec, & Trontelj, 1968) have described apparent retraining or reorganization of the central nervous system due to repetitive functional electrical stimulation, or FES as it is now known. Gracanin and Marincek (1970) reported long lasting effects from peroneal nerve activation in hemiplegic foot drop patients to the extent that many patients eventually walked without electrical stimulation. Waters et al. (1975) also reported permanent changes in walking patterns with positive changes in stride length,

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[1] Zankel (1960) reported successful treatment of hemiplegic upper limbs with stimulation assistive exercise (SAE) in which repetitive sinusoidal electrical stimulation of paralyzed muscle was done in conjunction with the patient's own volitional exercise effort.

single stance duration, and walking speed while Gracanin, Vrabic, and Vrabic (1976) showed positive changes in walking and posture in cerebral palsied children even after cessation of electrical stimulation. Vodovnik and Rebersek (1973) demonstrated greater voluntary torque production for dorsiflexion when stroke patients had volitional effort coupled with concurrent electrical stimulation. The latter study is quite similar to the stimulation assistive exercise (SAE) reported by Zankel in 1960 which coupled volitional exercise assisted by electrical stimulation with the result of improvement in range of motion of hemiplegic upper limbs.

Available evidence concerning the use of functional electrical stimulation as a technique for neurological retraining is not plentiful, and not without methodological deficiencies. Improvements cited previously, for example, could have resulted from improvements in muscle strength and endurance rather than from changes in the neural control mechanisms. In several of the functional electrical stimulation studies the methodology employed could be likened to proprioceptive stimulation techniques; i.e. superimposing an electrical stimulation upon a volitional contraction (Liberson et al., 1961; Vodovnik and Rebersek, 1973; Zankel, 1960) may have acted much like any other proprioceptive stimulation technique to enhance volitional muscular activity.

As it has been used to date, functional electrical stimulation has been administered either to activate a paralyzed muscle to produce action where none can be produced volitionally, or to provide additional stimulation to an active but weakened muscle. Improvement in neural control mechanisms, however, cannot be separated from improvements in muscle strength and endurance

because no measures of neural control mechanisms have ever been collected. Since neurological retraining is of more importance than changes in the peripheral muscle apparatus, there is an obvious need to develop a research methodology which can differentiate true changes in neural coordination mechanisms from changes in the periphery. Through no accident, planned research strategies have been developed which provide both the technique and the basic knowledge necessary to distinguish true changes in neural coordination mechanisms from changes in the peripheral muscle site.

Neuromotor Coordination Mechanisms

Muscular coordination involves the participation of many muscles that essentially act in cooperation with each other to apply force and produce movement, or in opposition to each other to inhibit force application and impede movement. In effect, the properly timed contraction of muscles at specified force levels involving the agonist, antagonist, and synergist muscles must be done in a sequential order for movement to even occur and for efficiency of movement to be possible. The initiation of a movement, control during the movement, and the ending of the movement all depend upon basic neuromotor control mechanisms. It is obvious, therefore, that the timing of force applications by involved muscle groups is an inherent feature of skilled, coordinated human performance.

One of the more persistent lines of investigation dealing with coordinated movement and application of muscle force has been that dealing with maximum speed of limb movement. Since the force required to cause movement of a limb is proportional to the distance travelled over time squared, $F = 2md/t^2$ or $F = ma$, it was believed that the greater the static

force (F) of a muscle, the greater the speed of limb movement which could be produced. When studied in humans, however, the maximum speed of limb movement bore no relationship to the maximum isoemtric strength of the agonist muscle group.

Although the study of maximum strength of an agonist muscle group and maximum speed of movement has gone on for several decades, the present status of our understanding of this basic coordinated movement pattern remains painfully inadequate. An enormous amount of literature exists in support of the untenable notion that human movement does not obey the basic principle of physics that F = ma since maximum strength of an agonist muscle group simply does not correlate with maximum speed of limb movement. Henry and Whitley (1960), Henry (1960, 1962), Clarke (1960), Smith (1961a, 1961b, 1969), Rasch (1963), and Nelson and Fahrney (1965), for example, all studied the speed of various limb movements and reported non-significant correlations with maximal strength of the agonist muscles involved in producing the movement.

Work in our laboratory has approached the problem differently and we are now able to show that agonist muscle strength does not correlate with maximum speed of movement because the neuromotor coordination mechanism has been overlooked by past researchers. Any attempt to understand the mechanism involved in fast, ballistic, coordinated movement must consider the sequential timing of activation of agonist and antagonist muscle groups which control the application of force and produce any resultant movement.

In any movement of a limb, the force exerted by the prime movers must be large enough to overcome the inertia of the limb and the force applied by the antagonists. With the prime movers in control, movement of the

limb occurs as characterized by displacement, acceleration, and velocity parameters. Limb movement decelerates when the force applied by the antagonist is large enough to overcome the force of the prime movers. Most biomechanical analyses of limb speed of movement have carefully measured acceleration, velocity, point of deceleration, and total movement time. Unfortunately, the biomechanical approach has seldom--if ever--sought an explanation of the motion being analyzed in terms of mechanisms. Traditional speed-strength investigations have limited themselves to the study of the relationship between maximum strength of agonist muscles and limb speed of movement parameters. Even more surprising, the sequential order of muscle activation--which in effect produces and controls movement--has been similarly overlooked.

As long ago as 1677 Descartes postulated that the interaction between muscles was regulated by a series of valves which adjusted the flow of animal spirits into flexor and extensor muscles. The question of actual interaction between agonist and antagonist muscles in humans has remained a key issue in the neurophysiology of movement ever since. Boulogne (1867) and Winslow (1732) believed that agonist and antagonist muscles contracted simultaneously while Bell (1823) and Pettigrew (1925) contended that the contraction of one muscle was accompanied by relaxation of its antagonist.

Later work by Beaunis (1889) and Demeny (1890) utilized kymographic records and supported simultaneous contraction of agonist and antagonist muscles. Sherrington (1906), however, promulgated his famous principle of reciprocal innervation and showed that contraction of an agonist was accompanied by inhibition of the antagonist in laboratory preparations.

Tilney and Pike (1925) studied many different actions in the upper limbs and were able to verify the principle of reciprocal innervation in voluntary human movement as they showed both agonist and antagonist active at the same site.

The first electromyographic investigation of the problem by Golla and Hettwer (1924) and later electromyographic studies by Stetson and Bouman (1935), Hudgins (1939), and Bierman and Ralston (1965) suggested that the type of movement being studied was important. Antagonist muscles were usually found to co-contract during slow controlled movements, and found not to co-contract during fast ballistic movements. Additional research on the interaction between agonist and antagonist muscles in voluntary movements supports, in general, the phenomeon of co-contraction and that a relationship exists between the speed at which a limb is moved and the sequential order of muscle firing patterns.

A better understanding of the neuromotor control mechanisms involved in limb speed of movement came about when the research methodology of biomechanical analysis of motion was combined with the research methodology of the neurophysiology of movement. The first comprehensive study assessing the neurophysiology and the biomechanics of limb movement was done in our laboratory as part of a Ph.D. dissertation (Lagassé, 1975). By monitoring the time of arrival of nervous impulses to the biceps and triceps muscles, the initiation of a rapid forearm flexion movement, velocity, acceleration, and total speed of movement, Lagassé was able to show that the sequential order of agonist-antagonist muscle activation was a significant predictor of speed of movement; the multiple R of .68 being the highest correlation

ever shown between limb speed of movement and a criterion measure related to a probable mechanism. The addition of acceleration time, which alone correlated with speed of movement r = .79, resulted in a multiple R of .90. Lagassé also showed that practice influenced the timing and coordination of the neuromuscular control mechanism, largely by a change in the agonist-antagonist sequential innervation pattern.

Individuals capable of the fastest speed of limb movement do not contract agonist and antagonist muscles simultaneously to the degree that slower subjects do. Marked co-contraction, therefore, characterizes poor neuromotor control while definite separation of agonist and antagonist muscle contraction patterns with a minimum of overlap characterizes skilled neuromotor control. Changes in the agonist-antagonist contraction pattern, furthermore, actually constitutes a pure measure of motor learning reflecting an inherent nervous system capability to learn which might very well be related to a number of other motor performance qualities including motor educability, the development of power, and the capacity to learn motoric skill tasks.

As part of a grant from the U. S. Army Medical Research and Development Command (DAMD 17-80-0101), we have been investigating the neuromotor coordination mechanisms involved in rapid limb movements. Mathematical models have been developed using a two compartment representation of the neuromuscular system involved in voluntary fast arm movements to a target. The first compartment accepts averaged biceps and triceps EMG signals as inputs, and models the arm's musculo-skeletal response by predicting angular position $Q(t)$ and velocity $\dot{\phi}(t)$ over the corresponding movement time. This is called the

$E/Q/\dot{\phi}$ (t) model, short for EMG/elbow torque = Q/elbow angular velocity $dQ/dt = \dot{\phi}$, and its defining equation is of the form:

$$\dot{\phi}\ (t) = (\text{Extensor torque } Q_E) - (\text{Flexor torque } Q_F).$$

The second compartment of the model, called the command/neural control/EMG or Com/Cont/E model, accepts the volitional commands as inputs, and models the nervous system response by producing a smoothed flexor and extensor EMG signals to feed into the $E/Q/\dot{\phi}$ model. Input commands are presumably via the pyramidal tract from the cerebellum to the brain-stem and spinal cord (cf. Miles and Evarts, 1979).

The purpose of the two compartment model is to separate various control functions in the ballistic arm movement system well enough to understand where adaptation occurs as speed, precision, and coordination changes occur due to practice. The appropriate connection of the two compartment model provides a complete model for fast limb movement as driven by volitional commands (Kilmer, Kroll, and Congdon, 1981).

Analysis of the empirical data on limb movement and parameters in the mathematical model describing neuromotor coordination mechanisms shows quite clearly that two major mechanisms are operative. The first mechanism involves the temporal sequence of agonist and antagonist firing patterns. If the agonist begins its contraction without co-contraction of the agonist, faster limb movement is produced. Antagonist muscle firing, however, is needed to allow precision of movement to a target. When the antagonist fires early so as to overlap with agonist firing, limb movement is slowed. If the antagonist fires later, thus allowing agonist contraction to manifest itself without competition, acceleration continues longer and

faster limb movement is produced. Indeed, one of the marked practice effects found is that antagonist firing occurs later during the movement.

The second major mechanism involves the ratio of flexor to extensor EMG intensity and duration. It is self-evident that the flexor EMG intensity and duration must be greater than comparable extensor EMG activity if a limb flexion movement is to be produced. As we have shown (Kilmer, Kroll, and Congdon, 1981; Kroll and Kilmer, 1981), this flexor/extensor EMG ratio is as critical as the temporal firing sequence of agonist and antagonist muscles in producing fast and efficient limb movement.

Thus, the temporal sequence of agonist and antagonist firing, and the flexor to extensor EMG ratio intensity and duration are the most critical neuromotor coordination mechanisms involved in limb movement speed. Although our analyses suggest the existence of other factors in limb movement, such as muscle fiber type composition and synchronized versus asynchronized motor unit firing patterns, the two major mechanisms identified and quantified are capable of excellent prediction of limb movement. The accuracy with which the mathematical model can predict limb movement can be appreciated by an inspection of Figure 1. In Figure 1, the solid line represents actual limb flexion displacement in a fast movement and the dotted circles are the predicted positions. As can be seen, predictions of model are extremely accurate.

Based upon results to date, it seems allowable to say that we understand a great deal about the major neuromotor coordination mechanisms involved in fast human limb movement. The mathematical

model described previously can predict the manner in which fast and efficient limb movement can be accomplished. As mentioned before, functional electrical stimulation (FES) techniques can be employed to stimulate human limbs in accordance with optimized models for efficient limb movement. The final issue is whether or not the blending of mathematical models of neuromotor coordination mechanisms with functional electrical stimulation techniques can produce real changes in limb movement patterns. That is, can functional electrical stimulation administered as prescribed by a mathematical model of neuromotor coordination produce any changes in the neuromotor coordination mechanisms controlling limb movement?

Motor Control and Learning Theories

The work of Lagasse and his associates have shown that functional electrical stimulation can produce improvements in limb movement patterns without any physical practice. That is, functional electrical stimulation produced learning via electrical stimulation alone. The importance of such "sensory imparted learning", or SIL, cannot be fully appreciated without some understanding of current theories of motor control and learning. Any attempt to present the numerous theories in an adequate manner would require a monumental effort, and is certainly not achievable in a concise manner. Such an effort is necessary, however, to provide some basis of reference against which to evaluate the significance of sensory imparted learning to motor control and learning theory.

At the present time there are numerous models and theories for motor control and learning ranging from simple descriptive taxonomies and hierarchical control models to highly technical information processing and cybernetic control models (Singer, 1980). In the context of motor

skill acquisition, however, there are two major categories into which current theories vying for acceptance can be classified. One category is the open-loop system of motor control and learning in which it is postulated that a motor skill is executed without being altered by any feedback or error detection mechanisms. Thus, a movement is programmed, and once started, the planned movement is unaffected by any peripheral feedback. Ballistic movements requiring less than one normal reaction time for execution are presumably under open-loop control. A second category is the closed-loop system in which feedback from the periphery can alter the motor task during its actual execution. The closed-loop system stipulates mechanisms involving feedback, error detection, and error correction during the actual execution of a movement task.

Open-loop proponents hold that a movement sequence obeys a response chaining principle in which each serial aspect of the movement is conditioned by the proprioceptive feedback of the preceding movement segment. Lashley (1917, 1951) was an early proponent of an open-loop theory and held that "...sensory factors play a minor part in regulating the intensity and duration of nervous discharge." The most important feature in open-loop theory is the central command mechanism while sensory feedback is viewed as relatively unimportant. Indeed, some open-loop proponents contend that sensory feedback is not even necessary. Taub and Berman (1960), for example, showed that monkeys could learn to squeeze a bulb in order to avoid a shock stimulus even when the involved limb was deafferentated. If feedback is necessary for learning, it may be provided by an efference copy or corollary discharge from the efferent command signal.

Closed-loop theories (e.g., Adams, 1971; Anokhin, 1969; Bernstein, 1967; Konorski, 1967; Sokolov, 1969; & Schmidt, 1975), on the other hand, place greater importance on concurrent feedback from an ongoing task. Schmidt (1975), for example, noted that "In order for the subject to be able to receive information about the correctness of the movement in relation to the desired outcome, he must be able to compare the actual feedback with the feedback expected." Keele and Summers (1976) likewise stated that "...the skill must be executed for learning to occur, for only in that way will kinesthetic feedback be available for conditioning to succeeding movements." In a paper proposing a model for sensorimotor control and learning, Raibert (1978) stated that "...active movement is essential to motor learning and sensorimotor adaptation." Raibert cited the classical work of Held (1961) and Hein and Held (1963) in support of his position.

Although this treatment of motor control and learning theory inadequately represents the actual complexity of the topic, it does allow one to point out the critical importance attached to the efferent signals from the central nervous system in the learning of a motor task. Whether viewed from open-loop or closed-loop schemas, active movement produced by efferent signals programmed by central command mechanisms is considered to be absolutely essential to motor skill acquisition. Present motor learning theories clearly make no provision for motor skill acquisition without efferent signals producing active movement.

Since sensory imparted learning (SIL) has been demonstrated several times, it seems reasonable to suggest that functional electrical stimulation applied in accordance with a model for neuromotor coordination mechanisms

for limb movement produces a sensory stimulus pattern acceptable to the central nervous system responsible for motor learning. The "language" of this central nervous system center obviously includes the relevant parameters of (1) temporal/sequential firing patterns of muscles involved in the movement synergy, and (2) the EMG ratio representing intensity of agonist and antagonist muscle contraction. Receipt of such relevant sensory stimulus patterns is followed by development of efferent motor programs which "match" the sensory stimulus patterns and eventually become available under volitional control. Thus, the term sensory imparted learning describes the sequence of such motor skill acquisition. For convenience, the term "reverse loop motor learning" seems to have merit as a description of the sensory imparted learning phenomenon in a theoretical context.

Sensory Imparted Learning

Several investigators reporting on the efficacy of functional electrical stimulation have noted relatively long lasting effects upon performance capabilities and have suggested such changes represent retraining or reorganization of the central nervous system (Gracanin and Marincek, 1970; Gracanin, Vrabic, and Vrabic, 1976; Waters, McNeal, and Perry, 1975; Vodovnick and Rebersek, 1973). Such reports of neurological retraining, however, cannot be said to be free of criticism. For example, observed improvements in functional capacities could have resulted from improved muscle strength and endurance rather than from actual changes in neural control mechanisms. In all of the studies reported, improvement in neural control mechanisms cannot be separated from improvements in muscle strength or endurance simply because no assessment of neural control mechanisms was ever made.

It should be obvious that the parameters described in the mathematical model of neuromotor coordination mechanisms responsible for control of limb movement represent a suitable set of criterion measures for assessing reorganization of the central nervous system or neurological retraining. Thus, we propose that any changes produced in these model parameters brought about by a therapeutic regimen can be interpreted as actual changes in the central nervous system separate from any changes produced in the peripheral musculature. Furthermore, the question of whether functional electrical stimulation can produce such changes has already been answered in several crucial investigations conducted by Pierre Lagassé at Laval University in Quebec City, Canada.

In a series of studies, functional electrical stimulation (FES) applied in accordance with neuromotor coordination principles has been shown capable of producing improvement in several physical performance tasks without benefit of actual physical practice. Fleury and Lagassé (1979), for example, demonstrated FES to be an effective means for improving total reaction time and its central nervous system component of premotor time. Actual muscle contraction time (or motor time in fractionated reaction time nomenclature) was the same in FES and physical practice groups of subjects. In a subsequent study, Lagassé, Boucher, Samson, and Jacques (1979) applied electrical stimulation to three different paired muscle groups involved in a weight lifting task, the clean and jerk lift. The muscle groups were stimulated in a temporal sequential order equivalent to that exhibited by a highly skilled record holder for the clean and jerk lift. Male subjects (N = 12) stimulated with an "optimized" muscle synergy pattern showed improvement without any physical practice, exhibiting acquisition of a proper motor

pattern for execution of the lifting task. Boucher and Lagassé (1981) later showed that maximum speed of a horizontal arm sweep task was significantly improved by functional electrical stimulation (FES), again based upon an optimized pattern of neuromotor coordination control mechanisms. Significant changes in limb velocity, acceleration, and temporal firing patterns of the three involved paired muscle groups were shown. Thus, functional electrical stimulation--with no actual physical practice--produced changes in the central nervous system. Motor learning was achieved strictly through a sensory pathway.

Sensory imparted learning (SIL) through use of functional electrical stimulation has been reported only once before, and even then some skepticism about the claims was apparent. Writing in Neural Organization and its Relevance to Prosthetics (1973), Reswick noted:

> It was also at about the same time in 1965 that L. A. Alyeyev and S. G. Bounimovich reported some interesting developments in electrical stimulation from the Institute of Cybernetics in Kiev. Alyeyev had developed a multichannel stimulating machine which he called the Miotone. He claimed it operated on the following principle: Myoelectric signals of muscles used in various functions previously recorded on magnetic tape obtained from 'donors' were used to modulate up to six channels of an electrical stimulating machine to produce stimulation signals which were controlled by the original EMG patterns. He claimed to be able to produce thereby functional movements in various types of paralysis in patients. These functional movements produced by electricity not only caused muscle hypertrophy and improved metabolic functions but also assisted in the reorganization of the central nervous system so that stroke patients were able to regain control of various paralyzed functions. To date no controlled study has been reported to confirm these assertions.

It seems allowable to suggest that Alyeyev and Bounimovich (1965) applied functional electrical stimulation to hemiplegic limbs in accordance with a crude model of neuromotor coordination mechanisms, using EMG patterns from "donors". The series of studies by Lagassé and his associates previously

cited used functional electrical stimulation based upon an improved model of neuromotor coordination mechanisms and successfully produced improvement without physical practice in several performance tasks. Lagassé and his associates, however, were using only an improved model of neuromotor coordination mechanisms, and not the best model. Although the neuromotor coordination mechanism of temporal sequence of muscle firing was controlled, the second major mechanism involving agonist to antagonist EMG duration and intensity was not described until quite recently (Kilmer, Kroll, and Congdon, 1981). Thus, if functional electrical stimulation is applied using the full and most modern model of neuromotor coordination mechanisms for control of limb movements, even greater effects can be expected.

Summary

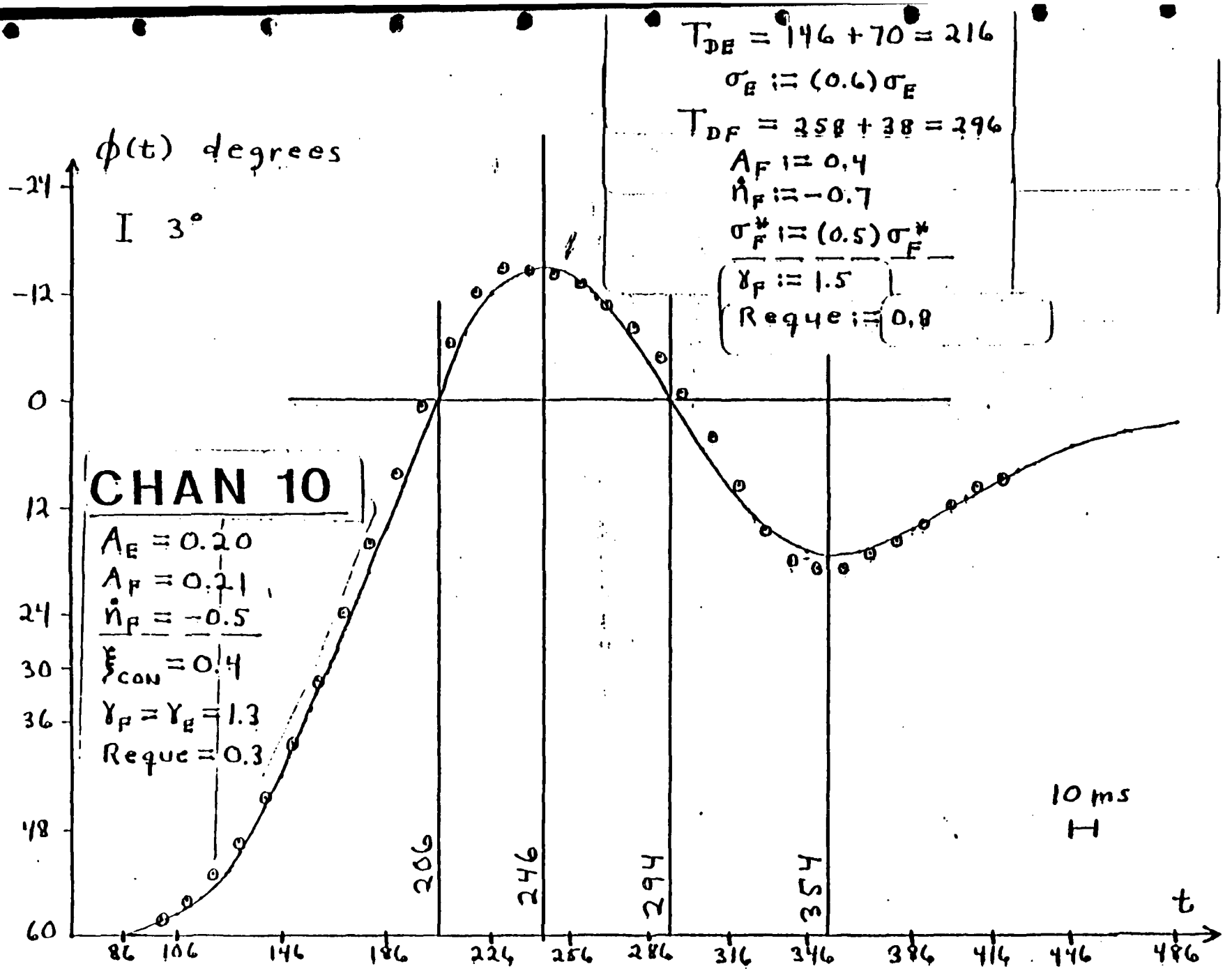
It has been proposed that the implicit goal of all neurophysiologically based therapeutic exercise systems is to transmit sensory stimulus patterns to the central nervous system which are more intense than that produced without benefit of proprioceptive facilitation. Implicit in such a treatment rationale is the belief that transmission of heightened sensory stimulus patterns to the central nervous system will enhance recovery of lost neurological functions. Various mechanisms for neurological function recovery have been proposed: collateral sprouting, unmasking, reactive synaptogenesis, sublimal fringe neuron activation, reclaiming of ipsilateral nerve pathway control tracts.

Motor command centers in the brain which have been damaged by some pathology can produce only inefficient and incorrect innervation commands

to the muscles. Limbs afflicted by some pathological condition can produce only inefficient and incorrect volitional movement patterns. Such inefficient and incorrect volitional movement produces a sensory stimulus pattern which is itself inefficient and incorrect, even if enhanced by proprioceptive facilitation techniques. If recovery of central nervous system functioning is dependent to any degree upon the quality of the sensory stimulus pattern received, the recovery is likely to be slow and dependent upon gradual improvement in the quality of the limb movement synergy which produces slight sensory stimulus patterns.

If, however, the sensory stimulus pattern transmitted to the central nervous system from an afflicted limb was not an impoverished pattern, but a sensory stimulus pattern representing optimally efficient limb movement, the likelihood of improved neurological recovery might be enhanced. Such a possibility now seems feasible because of developments in the areas of functional electrical stimulation, neuromotor coordination mechanisms controlling fast limb movements, and sensory imparted learning. These relatively distinct areas of research have an obvious relevance to each other, and a blending of their complementary components holds promise for a vastly improved rehabilitation regimen for neurological recovery. Based upon a mathematical model of neuromotor coordination mechanisms for optimally efficient limb movement, functional electrical stimulation can be applied to an afflicted limb which can produce an optimal sensory stimulus pattern for transmission to the central nervous system. If neurological recovery is dependent to any degree upon the quality of the sensory stimulus pattern, receipt of such optimized sensory stimulus patterns should be more effective than any present therapeutic exercise regimen. Since the functional electrical

stimulation is applied according to a mathematical model for neuromotor coordination mechanisms, a suitable set of criterion measures are available for assessing true neurological changes from changes in the peripheral musculature.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REFERENCES

Adams, J. A. A closed-loop theory of motor learning. Journal of Motor Behavior 3: 111-149, 1971.

Aleyeyev, L. S. and Bounimovich, S. G. On the question of control of movement of the anatomical-physiologic structure in normality and pathology. Modeling Techniques in Biology and Medicine, Institute of Cybernetics, Ukranian Academy of Science, Kiev, 1965, pp. 31-37.

Anokhin, P. K. Cybernetics and the integrative activity of the brain. In M. Cole & I. Maltzman (eds). A Handbook of Contemporary Soviet Psychology. New York: Basic Books, 1960. Pp. 830-856.

Ayres, A. J. Sensory Integration and Learning Disorders. Los Angeles: Western Psychological Services, 1973.

Bach-y-Rita, P. Central nervous system lesions: sprouting and unmasking in rehabilitation. Archives of Physical Medicine and Rehabilitation 62: 413-417, 1981.

Bach-y-Rita, P. Brain plasticity as basis for therapeutic procedures. In Bach-y-Rita, P. (ed): Recovery of function: Theoretical considerations for brain injury. Baltimore, University Park Press, 1980, pp. 225-263.

Baker, L., Yeh, C., Wilson, D. and Waters, R. L. Electrical stimulation of wrist and fingers for hemiplegic patients. Physical Therapy 59: 1495-1499, 1979.

Beaunis, H. Recherches physiologiques sur la contraction simultanee des muscles antagonistes. Archives de Physiologie et Pathologique 1: 110-151, 1889.

Bell, C. Anatomy and physiology of the human body. Philosophical Transactions 7: 110-151, 1823.

Bernstein, N. The co-ordination and regulation of movements. London: Pergamon Press, 1967.

Bierman, W. and Ralston, H. J. Electromyographic study during passive and active flexion and extension of the knee of the normal subject. Archives of Physical Medicine and Rehabilitation 46: 71-75, 1965.

Boucher, J. P. and P. P. Lagasse'. Effect of functional electrical stimulation (FES) on neuromuscular coordination mechanisms. Journal of Biomechanics 14: 498-499, 1981. (Abstract)

Bobath, B. Adult Hemiplegia: Evaluation and Treatment. London: W. Heinemann Medical Books, 1970.

Brindley, G. S., Polkey, C. E., and Rushton, D. N. Electrical splinting of the knee in paraplegia. Paraplegia 16: 428-435, 1978-79.

Brunnstrom, S. Movement Therapy in Hemiplegia. New York: Harper & Row, 1970.

Buell, S. J. and Coleman, D. P. Dendritic growth in aged human brain and failure of growth in senile dementia. Science 206: 854-856, 1976.

Carnstam, B., Larsson, L. E. and Prevec, T. S. Improvement of gait following functional electrical stimulation. Scandanavian Journal of Rehabilitation Medicine 9: 7-13, 1977.

Clarke, D. H. Correlation between the strength/mass ratio and the speed of an arm movement. Research Quarterly 31: 570-574, 1960.

Cotman, C. W. (ed). Neuronal plasticity. New York: Raven Press, 1978.

Cotman, C. W., Nieto-Sampedro, M. and Harris, E. W. Synapse replacement in the nervous system of adult vertebrates. Physiological Reviews 61: 684-784, 1981.

Delacato, C. H. Neurological Organization and Reading. Springfield, Illinois: C C Thomas, 1966.

Demeny, C. Du role mecanique des muscles antagonistes. Archives de Physiologie Normale et Pathologique 2: 747-752, 1890.

Descartes, R. L'Homme. Paris: Louis de la Forge, 1677.

Dimitrijevic, M. R., Gracanin, F., Prevec, T., and Trontelj, J. Electronic control of paralyzed muscles: neurophysiological considerations. Biomedical Engineering 3: 8-14, 1968.

Doman, R. J., Spitz, E. B., Zucman, E., Delacato, C. H., & Doman, G. J. Children with severe brain injuries: neurological organization in terms of mobility. JAMA 174: 257-262, 1960.

Eccles, J. C. Synaptic plasticity. Naturwissenschaften 66: 147-153, 1979.

Fay, T. The use of pathological and unlocking reflexes in the rehabilitation of spastics. American Journal of Physical Medicine 33: 347-352, 1954.

Flanagan, E. M. Methods for facilitation and inhibition of motor activity. American Journal of Physical Medicine 46: 1006-1011, 1967.

Fleury, M. and Lagassé, P. Influence of functional electrical stimulation training on premotor and motor reaction time. Perceptual and Motor Skills 48: 387-393, 1979.

Frenkel, H. S. The treatment of tabetic ataxia. Philadelphia: Blakiston, 1902.

Frostig, M. Movement Education: Theory and Practice. Chicago: Follett, 1970.

Golla, F. and Hettwer, J. A study of the electromyograms of voluntary movement. Brain 47: 57-69, 1924.

Gracanin, F. and Marincek, I. Long term effects of electrical stimulation. In M. M. Gavrilovic and A. B. Wilson, Jr., (eds). Advances in External

Control of Human Extremities. Belgrade: Yugoslav Committee for Electronics and Automation, 1970, pp. 495-501.

Gracanin, F., Vrabic, M. and Vrabic, G. Six years experience with FES method applied to children. Europa Medicophysica 12: 61-68, 1976.

Halpern, D. Therapeutic exercises for cerebral palsy. In J. V. Basmajian (ed). Therapeutic Exercise, third ed. Baltimore: Williams & Wilkins, 1978. Pp. 281-306.

Harris, F. A. Facilitation techniques in therapeutic exercise. In J. V. Basmajian (ed). Therapeutic Exercise. Third edition, Baltimore: Williams & Wilkins, 1978. pp. 93-137.

Held, R. Exposure-history as a factor in maintaining stability of perception and coordination. Journal of Nervous and Mental Disease 132: 26-32, 1961.

Held, R., and Hein, A. Movement-produced stimulation in the development of visually guided behavior. Journal of Comparative and Physiological Psychology 56: 872-876, 1963.

Henry, F. M. Factorial structure of individual differences in limb speed, reaction, and strength. Research Quarterly 33: 70-74, 1962.

Henry, F. M. Factorial structure of speed and static strength in a lateral arm movement. Research Quarterly 31: 440-448, 1960.

Henry, F. M. and Rogers, D. E. Increased response latency for complicated movements and a "memory drum" theory of neuromotor reaction. Research Quarterly 31: 448-458, 1960.

Henry, F. M. and Whitley, J. D. Relationships between individual differences in strength, speed, and mass in arm movement. Research Quarterly 31: 24-33, 1960.

Hirt, S. Historical basis for therapeutic exercise. American Journal of Physical Medicine 46: 32-38. 1967.

Hudgins, C. V. The incidence of muscular contractions in reciprocal movements under conditions of changing loads. Journal of General Psychology 20: 327-338, 1939.

Jones, A. M. The traditional method of treatment of the cerebral palsied child. American Journal of Physical Medicine 46: 1024-1031, 1967.

Kabat, H. Proprioceptive facilitation in therapeutic exercise. In S. Licht (ed), Therapeutic Exercise, pp. 327-343, Baltimore: Waverly Press, 1965. Second Edition.

Keele, S. W. and Summers, J. J. The structure of motor programs. In G. E. Stelmach (ed). Motor Control: Issues and Trends. New York: Academic Press, 1976, pp. 109-142.

Kilmer, W., Kroll, W., and Congdon, V. An EMG-level muscle model for a fast arm movement to target. Biological Cybernetics (in press), 1981.

Knott, M. & Voss, D. Proprioceptive Neuromuscular Facilitation. New York: Hoeber, 1962.

Konorski, J. Integrative Activity of the Brain. Chicago: University of Chicago Press, 1967.

Lagassé, P. The prediction of maximum speed of human movement by two selected muscular coordination mechanisms and by maximum static strength and the effects of practice and fatigue upon maximum speed of human movement mechanisms. Unpublished Ph.D. dissertation. Amherst: University of Massachusetts, 1975. 159 pp.

Lagassé, P., Boucher, J., Samson, J. and Jacques, L. Training effects of functional electrical stimulation in weight lifting. Journal of Human Movement Studies 5: 61-67, 1979.

Lashley, K. S. The problem of serial order in behavior. In L. A. Jeffress (ed). Cerebral Mechanisms in Behavior. New York: Wiley, 1951. Pp. 112-136.

Lashley, K. S. The accuracy of movement in the absence of excitation from the moving organ. American Journal of Physiology 43: 169-194, 1917.

Lazarus, P. Die bahnungstherapie der hemiplegie. Zeitschrift fur Klinische Medizin 45: 314-339, 1902.

Liberson, W. T., Holmquest, H. J., Scot, D. and Dow, M. Functional electrotherapy: stimulation of the peroneal nerve synchronized with the swing phase of the gait of hemiplegic patients. Archives of Physical Medicine and Rehabilitation 42: 101-105, 1961.

Licht, S. History of electrotherapy. In S. Licht (ed). Therapeutic Electricity and Ultraviolet Radiation. New Haven, Connecticut: Licht, 1959, pp. 1-69.

McNeal, D. R. 2000 years of electrical stimulation. In T. T. Hambrecht and J. B. Reswick (eds). Functional Electrical Stimulation. New York: Dekker, 1977. Pp. 3-35.

Miles, F. A. and Evarts, E. V. Concepts of motor organization. Annual Review of Physiology 30: 327-362, 1979.

Nelson, R. C. and Fahrney, R. A. Relationship between strength and speed of elbow flexion. Research Quarterly 36: 455-463, 1965.

Raibert, M. H. A model for sensorimotor control and learning. Biological Cybernetics 29: 29-36, 1978.

Rasch, P. J. Relationship of arm strength, weight, and length to speed of arm movement. Research Quarterly 23: 328-332, 1963.

Reswick, J. B. A brief history of functional electrical stimulation. In W. S. Fields and L. A. Levitt (eds). Neural Organization and Its Relevance to Prosthetics. New York: International Medical Books, 1973, pp. 3-15.

Rood, M. S. Neurophysiological reactions as a basis for physical therapy. Physical Therapy Review 34: 444-449, 1954.

Schmidt, R. A. A schema theory of discrete motor skill learning. Psychological Review 82: 225-260, 1975.

Sherrington, C. S. Integrative Action of the Nervous System. New York: Yale University Press, 1906.

Singer, R. N. Motor Learning and Human Performance. Third ed. New York: Macmillan, 1980.

Smith, L. E. Individual differences in strength, reaction latency, mass and length of limbs, and their relation to maximal speed of movement. Research Quarterly 32: 208-220, 1961a.

Smith, L. E. Relationship between explosive leg strength and performance in the vertical jump. Research Quarterly 32: 405-408, 1961b.

Smith, L. E. Specificity of individual differences of relationship between strength and speed of forearm flexion. Research Quarterly 40: 191-197, 1969.

Sokolov, E. N. The modeling properties of the nervous system. In M. Cole & I. Maltzman (eds). A Handbook of Contemporary Soviet Psychology. New York: Basic Books, 1969. Pp. 671-704.

Stelmach, G. E. (ed). Motor control: issues and trends. New York: Academic Press, 1976.

Stetson, R. H. and Bouman, H. D. The coordination of simple skilled movements. Archives Neerlandaises de Physiologie de l'homme et des Animaux 20: 177-254, 1935.

Swenson, J. R. Therapeutic exercise in hemiplegia. In J. V. Basmajian (ed). Therapeutic Exercise, third ed. Baltimore: Williams & Wilkins, 1978. Pp. 325-348.

Taub, E. and Berman, A. J. Movement and learning in the absence of sensory feedback. In S. J. Freedman (ed). The Neuropsychology of Spatially Oriented Behavior. Homewood, Ill.: Dorsey Press, 1968.

Tilney, F. and Pike, F. H. Muscular coordination experimentally studied in its relation to the cerebellum. Archives of Neurology and Psychiatry 13: 289-334, 1925.

Vodovnik, L. and Rebersek, S. Improvement in voluntary control of paretic muscles due to electrical stimulation. In W. S. Fields and L. A. Levitt (eds). Neural organization and its relevance to prosthetics. New York: International Medical Books, 1973.

Wall, P. D. Mechanisms of plasticity of connection following damage in adult mammalian nervous systems. In P. Bach-y-Rita (ed), Recovery of Function: Theoretical Considerations for Brain Injury Rehabilitation. Baltimore: University Park Press, 1980, pp. 91-105.

Waters, R. L., McNeal, D. and Perry, J. Experimental correction of footdrop by electrical stimulation of the peroneal nerve. Journal of Bone and Joint Surgery 57: 1047-1054, 1975.

Zankel, H. T. Stimulation assistive exercise in hemiplegia. Geriatrics 15: 616-622, 1960.

APPENDIX D

ARTICLE

PREDICTION OF MALE AND FEMALE ISOMETRIC ARM STRENGTH BY ANTHROPOMETRIC MEASURES

PREDICTION OF MALE AND FEMALE ISOMETRIC ARM STRENGTH BY ANTHROPOMETRIC MEASURES*

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ABSTRACT

Twenty male and twenty female subjects (ages 17 to 28 years) were measured for maximal isometric elbow flexion and extension strength, upper limb volume, lengths and girths. Female flexion strength was 54.3% and extension strength 58.2% of males. Covariance adjustment for body weight and upper limb volume increased female flexion strength to 78% and extension strength to 73% of males, but males were still significantly stronger. Arm girths and limb volume exhibited larger sex differences than strength measures. Limb lengths were not as important as girths; and body weight and limb volume importance in strength prediction differed in males and females. Multiple R's of .84 to .94 for strength resulted from a full set of anthropometric predictors. Simple measures of limb girths and lengths, with and without body weight, were as good or better than segmental limb volumes in strength prediction. It was clear that: (1) the importance of anthropometric measures as strength predictors differs not only between the sexes but between strength measures as well; (2) male and female differences extend beyond absolute differences in anthropometric measures and includes differences in the relationship of upper limb anthropometric measures; (3) body size factors do not explain adequately the strength superiority of males.

Composite strength indices based upon representative batteries of strength measures have generally shown moderate but significant correlations with various physique measures (Jones, 1947; Laubach & McConville, 1966, 1969; Sills & Everett, 1953). The most common anthropometric measure assessed in conjunction with strength is undoubtedly body weight followed in frequency of usage by limb girths and lengths. Because of the accepted relationship between muscle cross-sectional area and strength, the use of anthropometric measures in strength studies seems logical. Unfortunately, much of the research dealing with the relationship of strength and anthropometric measures has been equivocal with many investigators questioning the usefulness of anthropometric measures in strength prediction (Cureton, 1947; Rasch & Pierson, 1963; Smith & Royce, 1963).

Only a few studies have included both males and females making it difficult to compare the importance of anthropometric predictors of strength between the sexes. Even fewer studies have included segmental limb volumes specific to the muscle group being studied. Due to provisions of the Equal Employment Opportunities Act, the increased presence of females in the military, and the growth of organized athletics for females, renewed attention has been given to differences and similarities of strength and related parameters in females and males. In the present study, equal numbers of males (N=20) and females (N=20) were measured for maximal isometric strength of elbow flexion and extension. Strength measures were then correlated by sex with body weight, limb lengths and girths, and segmental upper limb volumes. As will be shown, the importance and effectiveness of anthropometric measures as strength predictors differs not only between the sexes but between strength measures as well.

MATERIALS AND METHODS

Twenty male and twenty female subjects (ages 17 to 28 years) were given medical clearance and signed informed consent forms as prescribed by university guidelines for the protection of human subjects. Each subject completed three data collection sessions within a one-week period. Half of the subjects continued on in a longer study of arm movement speed to be reported elsewhere.

Anthropometric measures. In addition to body weight, segmental limb volumes for the right upper arm, forearm, and hand were determined using a water displacement technique (Dempster, 1955; Katch & Weltman, 1975). Segmental volumes were also estimated by an anthropometric technique (Katch & Weltman, 1975; Sady et al., 1978). Measures of limb lengths and girths required for volume estimation, and employed as separate predictors of strength, were as follows.

Lengths

L1	acromion process to deltoid tubercle
L2	deltoid tubercle to olecranon process
L3	olecranon process to styloid process of ulna
L4	styloid process of ulna to tip of third finger

Girths

AC	circumference at acromion process
DEL	circumference at deltoid tubercle
ELB	circumference at olecranon process
WJS	circumference at distal space to styloid process of ulna (wrist joint space)
HND	thickness of base of hand, cross-section height thenar eminence and hypothenar eminence

Isometric strength. Four 5-sec maximal isometric contractions were secured for right elbow flexion and extension strength. Each trial was separated by a 1-minute rest interval and the flexion-extension series by a 7-10 minute rest interval. The order of flexion and extension testing was

balanced over subjects with no differences in mean strength due to order of testing being detected. Two trials were maximal voluntary contractions (MVC) measures where subjects were instructed to exert a maximal contraction for 5-seconds. The other two trials were fast MVC (FMVC) measures where subjects were instructed to reach maximum tension as quickly as possible and maintain maximum until commanded to stop. Peak tension output of each trial was taken as maximum strength.

Subjects were tested in a sitting position, the chest braced against a padded table support, with a wide nylon belt around the upper body to prevent movement away from the test apparatus. The upper arm was parallel to, and supported by, the table top at a 90 degree angle to the shoulder. The forearm was positioned at angles of 75 degrees for flexion and at 90 degrees for extension. In accordance with Basmajian and Latif (1957), the elbow angles are given as the compliment of the angle between the forearm and the upper arm. A wrist cuff connected via an adjustable steel cable to a Statham strain gauge which monitored tension output and was recorded on a Beckman Type R Dynagraph. The angle of pull for all trials was at 90 degrees to the strain gauge with the hand positioned midway between supination and pronation. Forearm position is known to affect the expression of elbow strength but the use of a wrist cuff effectively eliminates such effects (Provins & Salter, 1955). Alteration in forearm position from the midline during maximal efforts would thus not affect the expression of maximum strength.

TABLE 1 TO APPEAR ABOUT HERE

Table 1

Mean Values for Isometric Strength (KG)

	Male (N=20)		Female (N=20)	
	Mean	SD	Mean	SD
Flexion				
MVC	21.3	4.7	11.6	3.2
FMVC	21.1	4.4	11.6	2.9
Extension				
MVC	17.7	3.5	10.3	3.3
FMVC	17.3	4.1	10.6	3.6

RESULTS

As seen in Table 1, maximum isometric elbow flexion strength was higher than extension strength by 20% in males and 12.1% in females. The superiority of elbow flexion strength is a common finding in males (Knapik & Ramos, 1980; Singh & Karpovich, 1968), in females (Kroll, 1972), and in elite, trained weight lifters and middle distance runners (Tornvall, 1963). Higher elbow flexion strength prevails even though the brachial triceps typically possesses a higher percentage of fast-twitch muscle fibers (Buchthal & Schmalbruch, 1970; Johnson et al., 1973), and has a larger physiological cross-sectional area (An et al., 1981). One factor enabling the elbow flexors to produce higher strength is a decided mechanical advantage. The major flexors of the forearm (biceps brachii, brachialis, and brachioradialis) have force arms two to four times longer than the brachial triceps (Ikai & Fukunaga, 1968; Morris, 1948; Wilkie, 1950).

Males were significantly stronger than females on both elbow flexion and elbow extension isometric strength as other investigators have reported (Petrofsky & Phillips, 1980; Singh & Karpovich, 1968). Female flexion strength was 54.3% and extension strength 58.2% when expressed as a percentage of male strength. Hettinger (1961) reported female elbow flexion and extension strength as being 55% of males, which is comparable to the present results, while Singh and Karpovich (1968) reported smaller values of 48% (extension) and 44% (flexion). Nordgren (1972) found female elbow flexion strength to be 54% and extension strength 52% of male strength.

There were no significant differences between MVC and FMVC strength in either males or females. Observed differences were less than one kg with

males exhibiting higher MVC and females higher FMVC values. Caldwell et al. (1974) and Rupp and Baranowski (1934) also reported no difference between slow and fast expression of maximum grip strength. In elderly males, however, a fast maximal voluntary contraction produces a significantly higher isometric strength by some 19% compared with a gradual expression of isometric knee extension strength (Clarkson et al., 1981). MVC and FMVC strength correlated highly in males and females with r's ranging from .88 to .98. Since there were no differences between MVC and FMVC absolute values, and since the measures were so highly correlated, all further data analysis reports only the MVC results.

TABLE 2 ABOUT HERE

Anthropometric measures. As seen in Table 2, males and females differed significantly on all anthropometric measures except L1 (acromion process to deltoid tubercle) and L3 (olecranon process to styloid process of ulna). Males were 14.1 kg or 23.5% heavier than females in body weight while total upper limb volume was 1,191.8 ml or 43.9% larger in males. Differences in arm girths ranged from 13.6% to 18.2% and in limb lengths from 5.25% to 10.9% in favor of the males. Thus, arm girths and limb volume exhibited larger sex differences than limb strength measures.

In a study involving 41 adult males Roberts et al. (1959) reported that upper arm girth correlated negatively with four longitudinal measures (height, upper arm length, forearm length, and hand length). The same general relationship is seen for males in the present study (see Table 4) with AC girth (circumference at acromion process) and DEL girth (circumference at deltoid

Table 2

Means for Body Weight (kg), Girths (cm), Length (cm), and Limb Volume (ml)

	Male (N=20)		Female (N=20)	
	Mean	SD	Mean	SD
Body Weight	74.0	6.1	59.9	7.8
Girths				
AC*	41.0	2.8	34.7	2.5
DEL*	29.6	2.4	25.9	2.0
ELB*	27.8	1.3	23.7	1.2
WJS*	17.6	0.6	15.4	0.6
HND*	5.0	0.3	4.4	0.5
Length				
L1	14.4	2.1	13.5	1.5
L2*	15.2	1.0	13.7	1.1
L3	24.3	2.3	23.1	1.6
L4*	21.0	0.9	19.3	0.9
Limb Volumes				
SEG1*	1443.3	292.1	1012.6	224.3
SEG2*	1003.5	160.4	682.4	121.1
SEG3*	1034.5	106.9	718.1	106.5
SEG4*	428.4	61.5	304.8	41.0
Total*	3909.7	479.1	2717.9	435.9

*Significant difference between males and females, $p < .01$.

tubercle) each failing to correlate significantly with any of the four limb length measures. In females, however, five out of the eight possible correlations between these two girth measures and the four limb length measures were significant. Differences between males and females thus extend beyond absolute differences in anthropometric measures and includes differences in the relationship of anthropometric measures in the upper limb as well.

TABLE 3 ABOUT HERE

Observed strength differences between men and women are presumed to be strongly influenced by over-all body size rather than by muscle quality. Comparison of male and female strength, therefore, is more prudent if body size factors can be controlled. In the present study, both body weight and total upper limb volume demonstrated significant relationships with strength but the relationship was different in the two sexes. A covariance analysis was therefore made to control for the influence of both body weight and upper limb volume upon strength. The adjusted means for male and female elbow flexion and extension strength are presented in Table 3. The effect of covariance adjustment for body size is best observed when female strength is expressed as a percentage of male strength. Initially female flexion and extension strength was 54.3% and 58.2% of male strength. Adjusted for body weight and upper limb volume, female flexion and extension strength increased to 78% and 73% of male strength. In agreement with others (Hoffman et al., 1979; Morrow & Hosler, 1981), however, male strength was still superior even after covariance adjustment for body size.

Table 3

Adjusted Means for Flexion and Extension Strength in Males and Females

	Male (N=20)	Female (N=20)	% Strength (Female/Male)
Flexion			
Raw	47.00	25.50	54%
Adjusted	40.51	31.90	78%
Extension			
Raw	39.10	22.70	58%
Adjusted	35.60	26.30	73%

TABLE 4 ABOUT HERE

Inter-correlation matrix. Total upper limb volume in females correlated significantly with all other variables (see Table 4). In males, however, total upper limb volume failed to correlate with body weight (r=.35, ns), hand girth (r=.24, ns), length 3-forearm (r=-.04, ns), or with any of the strength measures (r's from .05 to .34, ns). Body weight correlated significantly with MVC flexion strength in males (r=.69, $p < .01$) and in females (r=.59, $p < .01$) but failed to correlate with MVC extension strength in either sex. Girth measures had more and higher correlations with strength measures in females than in males. Of the eight possible correlation coefficients for limb length with strength measures, two correlations were significant in females (both with flexion strength), and only one correlation was significant in males. Limb length 3 (forearm) and length 4 (hand) correlated with MVC flexion strength (r=.60 and .71) in females while in males these limb lengths showed no relationship with strength. The general impression given by the correlation matrix suggests that limb lengths are not as important as limb girths, and the importance of body weight and limb volume differs between males and females in the prediction of elbow flexion and extension strength.

Multiple correlation analysis. A better understanding of the relative importance of anthropometric measures in the prediction of isometric elbow flexion and extension strength can be realized by multiple correlation analysis. Using stepwise multiple correlation, analysis was made in a sequential stage-wise fashion with each stage including a different set or combination of anthropometric predictors. In the first stage, body weight (BWT) and total upper limb

Table 4

Correlation Matrix[a]

	BW	AC	DEL	ELB	WJS	HND	L1	L2	L3	L4	S1	S2	S3	S4	MVC^{EX}	MVC^{FL}	VTOT
Body Weight (BW)	-	.52*	.69**	.65**	.73**	.46*	.24	.56**	.69**	.65**	.58**	.82**	.81**	.54*	.35	.59**	.77**
Acromion Girth (AC)	.24	-	.57**	.46*	.55*	.38	.53*	.37	.57**	.63**	.86**	.60**	.61**	.44*	.36	.61**	.80**
Deltoid Girth (DEL)	.53*	.51*	-	.75**	.74**	.45*	.32	.29	.57**	.45*	.69**	.83**	.80**	.53*	.60**	.59**	.83**
Elbow Girth (ELB)	.53*	.47*	.78**	-	.78**	.49*	.25	.27	.42	.49*	.55*	.78	.85**	.79	.80**	.73**	.78**
Wrist Girth (WJS)	.35	.28	.62**	.72**	-	.54*	.18	.26	.54*	.55*	.56	.71	.84**	.79	.49*	.57**	.77**
Hand Girth (HND)	.14	.38	.44*	.40	.21	-	.47*	-.005	.59	.67**	.54*	.33	.65**	.49*	.54*	.61**	.58**
Length 1 (L1)	-.28	.31	-.18	-.02	.12	-.15	-	.06	.48*	.17	.84**	.26	.40	.12	.24	.17	.61**
Length 2 (L2)	.41	-.02	.35	.51*	.44*	-.06	-.02	-	.49*	.49*	.29	.73**	.42	.30	.04	.38	.49*
Length 3 (L3)	.23	.08	.13	-.09	-.01	-.34	-.32	.01	-	.62**	.68**	.67**	.82**	.51*	.35	.60**	.79**
Length 4 (L4)	.10	.37	.18	.17	.26	.08	.27	.45*	.18	-	.49*	.59**	.65**	.62**	.37	.71**	.64**
Segment 1 Volume	.05	.79**	.37	.39	.38	.18	.80**	.06	-.15	.39	-	.64**	.72**	.41	.45*	.53*	.91**
Segment 2 Volume	.59**	.36	.85**	.88**	.69**	.28	-.12	.76**	.05	.32	.29	-	.85**	.64	.54*	.70**	.88**
Segment 3 Volume	.46	.32	.70**	.79**	.79**	.03	.02	.67**	.14	.43	.34	.86	-	.79	.67**	.78**	.92**
Segment 4 Volume	.15	.48	.14	.26	.55*	.22	.32	.27	.01	.46*	.48*	.24	.32	-	.57**	.64**	.68**
MVC Extension	.24	.01	.56**	.22	.16	.54*	-.34	.10	-.04	-.12	-.07	.37	.09	-.02	-	.82**	.60**
MVC Flexion	.69**	.23	.68**	.49*	.26	.41	-.38	.45*	.14	.32	.01	.66	.41	.10	.55**	-	.72**
Total Limb Volume	.35	.73**	.68**	.74**	.71**	.24	.49*	.48*	-.04	.50*	.84**	.73	.76**	.57	.10	.34	-

[a]Values below diagonal for males; values above diagonal for females.

*$P < .05$

**$p < .01$

volume (VTOT) were the only predictors. Next, body weight and segmental limb volumes were used as predictors followed by segmental limb volumes and total upper limb volume. Limb lengths and girths, with and without body weight and segmental limb volumes, were used as predictor sets to contrast the predictive efficiency of simple length and girth measures with limb volume and body weight measures. The final analysis used the full set of anthropometric measures to predict elbow flexion and extension MVC strength in males and in females. Results of the multiple correlation analyses are presented in Table 5. The decision rule for the inclusion of a predictor was that it must increase R^2 by at least five percent or that deletion must not decrease R^2 by more than five percent.

TABLE 5 ABOUT HERE

Limb volume would logically be expected to correlate more highly with limb strength than the more global anthropometric measure of body weight. Given the two predictors of body weight (BWT) and total upper limb volume (VTOT), however, BWT is a better predictor of male flexion strength ($r=.69$, $p < .01$) and extension strength ($r=.24$, ns). In females, however, VTOT is a better predictor than BWT for flexion strength ($r=.72$, $p < .01$) and extension strength ($r=.63$, $p < .01$). All subsequent multiple correlation analyses produced higher R's than the above which used only BWT and VTOT.

Multiple correlations using BWT plus segmental limb volumes were as good or better than prediction equations employing segmental limb volumes plus VTOT. In both sets of equations elbow flexion strength was better predicted than extension strength. Only limb volume segment 2 (deltoid tubercle to elbow)

Table 5

Multiple Correlation Analysis*

Body Weight (BWT) + Volume Total (VTOT)	R
Male	
Flexion BWT (47.6%)	.69
Extension BWT (5.8%)	.24
Female	
Flexion VTOT (51.8%)	.72
Extension VTOT (36.1%) + BWT (36%)	.60
BWT + Limb Volumes	
Male	
Flexion BWT (47.4%) + SEG2 (10.1%) + SEG3 (7.7%)	.81
Extension SEG2 (13.7%) + SEG3 (20.2%)	.58
Female	
Flexion SEG3 (60.1%)	.78
Extension SEG3 (45.1%) + BWT (11.0%)	.75
Limb Volumes + VTOT	
Male	
Flexion SEG2 (44.1%) + SEG3 (10.3%)	.74
Extension SEG2 (13.7%) + SEG3 (20.2%)	.58
Female	
Flexion SEG3 (60.1%)	.78
Extension SEG3 (45.1%)	.67

Limb Lengths + Limb Girths	R
Males	
Flexion DEL-G (46.3) + L1 (6.8) + L4 (8.8)	.79
Extension DEL-G (31.4) + ELB-G (11.8) + HND-G (13.0) + AC-G (13.1)	.83
Females	
Flexion ELB-G (53.1) + L4 (16.4)	.83
Extension ELB-G (64.6) + WJS-G (4.8) + HND-G (5.5)	.87
Limb Lengths + Limb Girths + BWT	
Males	
Flexion BWT (47.4) + DEL-G (13.8) + WJS-G (4.6) + L4 (6.0) + AC-G (5.3) + HND-G (4.1)	.90
Extension DEL-G (31.4) + ELB-G (11.8) + HND-G (13.0) + AC-G (13.1)	.83
Females	
Flexion ELB-G (53.1) + L4 (16.4)	.83
Extension ELB-G (64.6) + BWT (5.5) + HND-G (4.8)	.87
Limb Lengths + Limb Girths + Segmental Volumes + VTOT	
Males	
Flexion DEL-G (46.3) + L1 (6.8) + L4 (8.8) + SEG3 (3.1) + L2 (7.4)	.85
Extension DEL-G (31.4) + SEG3 (18.2) + AC-G (13.0) + SEG4 (7.7)	.84
Females	
Flexion SEG3 (60.1) + ELB-G (3.1) + WJS-G (4.0) + L1 (2.2) + AC-G (5.2) + HND-G (7.8)	.94
Extension ELB-G (64.6) + WJS-G (4.8) + HND-G (5.5)	.87

All Predictors

Males

Flexion BWT (47.4) + DEL-G (13.8) + WJS-G (4.6) +
L4 (5.9) + SEG1 (5.5) .88

Extension DEL-G (31.4) + SEG3 (18.2) + AC-G (13.0) +
SEG4 (7.7) .84

Females

Flexion SEG3 (60.1) + ELB-G (3.1) + WJS-G (4.0) +
L1 (2.2) + AC-G (5.2) + HND-G (7.7) .94

Extension ELB-G (64.5%) + BWT (5.5%) + HND-G (4.8%) .87

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*Values in parentheses indicate contribution to R^2.

and limb volume segment 3 (forearm) were selected from the volume predictors. BWT was added to SEG 2 and/or SEG 3 predictor sets raising the multiple R from .74 to .81 for male flexion strength and from .67 to .75 for female extension strength.

Limb length and girth predictor sets produced generally higher multiple R's than predictor sets employing segmental limb volumes, VTOT, and BWT. All extension strength predictors in males and females were girth measures while prediction equations for flexion strength had fewer predictors and contained both limb length and limb girth measures. Unlike prediction equations using segmental limb volumes, prediction equations using limb lengths and girths produced higher multiple R's for extension than for flexion strength. The addition of BWT to limb length and girth predictor sets improved the multiple R only for male flexion strength, r increases from .79 to .90.

When all predictors are included in the multiple R analysis (BWT, limb lengths and girths, segmental limb volumes, and total upper limb volume), the multiple R for male flexion strength containing five predictors of .88 is actually less than the mutiple R of .90 based upon six predictors without segmental limb volumes. For male extension strength, a multiple R of .84 with four predictors including segment limb volumes is quite similar to the multiple R of .83 based upon four limb girth predictors. A similar situation exists for the females. Regardless of whether segment limb volumes are included in the regression analysis, the same three limb girth measures are chosen for extension strength prediction. A six measure prediction equation including segmental limb volumes produces a multiple R of .94 for female flexion strength while a six measure prediction equation including only limb lengths, girths, and BWT produces a multiple R of .90.

DISCUSSION

The present results agree with many previous reports (Borchardt, 1968; Petrofsky & Lind, 1975; Rasch & Pierson, 1963; Royce, 1958; Smith & Royce, 1963) which have failed to show a consistent relationship between body weight and isometric strength. The present study showed a significant correlation between BWT and MVC flexion strength in both males ($r=.69$, $p<.01$) and females ($r=.59$, $p<.01$) but BWT failed to correlate with MVC elbow extension strength in either sex. Body weight correlated significantly with total upper limb volume (VTOT) in females ($r=.77$, $p<.01$) but not in males ($r=.35$, ns). VTOT correlated significantly with female elbow flexion strength ($r=.72$, $p<.01$) and female extension strength ($r=.62$, $p<.01$), but not with male strength measures. Thus, the relative importance of body weight and total upper limb volume as strength predictors differed between males and females and between elbow flexion and extension strength.

In agreement with others (Laubach & McConville, 1966; Tornvall, 1963), limb girth measures were better than limb lengths as strength predictors. However, this generalization requires some qualification. Although it is true that no limb length measures appeared in any prediction equations for elbow extension strength, several limb length measures did appear in elbow flexion strength prediction equations. The appearance of L4 (hand length) as a significant predictor of flexion strength in several equations is of interest. Roberts et al. (1959) also found that hand length correlated significantly with male flexion strength ($r=.59$) and extension strength ($r=.42$). Hand length (L4) appeared in six of the 16 multiple correlation analyses using limb girths and lengths as part of the predictor set. In every instance hand length appeared in prediction of flexion strength (four times for males,

twice for females), but never in prediction equations for extension strength. Since all strength measures were secured using a wrist cuff, the hand could not participate in, or contribute to, the actual expression of strength.

The importance of hand length in elbow flexion strength prediction equations could reflect some inherent feature of skeletal size, lever length, or somatotype related to isometric strength. The fact that hand length did not appear in any extension strength prediction equations, however, weakens support for such a speculation. In addition, body weight correlated significantly only with flexion strength in both males and females. Coupled with the finding of flexion over extension strength superiority in both sexes, the importance of hand length may be related to a recent observation by Goslin and Charteris (1982) concerning the arboreal heritage of humans. According to these authors, flexion strength superiority would be logical in arboreal locomotion where the elbow is chiefly used for suspension. On similar phylogenetic grounds, flexion strength superiority would be advantageously served by a longer hand for climbing and arboreal locomotion.

The absence of limb length measures in elbow extension strength prediction equations was also reported by Roberts et al. (1959) and may be due to anatomical features of the triceps brachii muscle. It is known that the three triceps brachii components have a larger volume but a shorter fiber length as compared to the biceps brachii muscle (An et al. 1981). Being a bipennate muscle with shorter and more obliquely running fibers, the triceps girth measure would logically be more important than length measures. The biceps brachii muscle, on the other hand, is a fusiform muscle with its fibers extending the entire muscle length, and one might expect limb length measures to be of more importance in the expression of elbow flexion strength.

In a study involving 41 adult males, Roberts et al. (1959) found that the multiple R's were slightly but consistently lower for prediction of elbow flexion than for elbow extension strength. No such consistent pattern in the present study was found although prediction equations for elbow flexion strength generally contained a greater number of predictor items than extension strength. Although Roberts et al. (1959) measured height, weight, three limb lengths, and two limb girths, their multiple correlation analyses reported only predictor sets of three measures; two of the three measures were always height and weight. The highest multiple R's they reported for prediction of elbow flexion (R=.73) and extension strength (R=.76) were lower than all multiple R's obtained in the present study which included body weight and limb lengths and girths as part of the predictor set. The multiple R's in the present study, ranging from .84 to .94, for the full predictor set analyses, are the highest multiple R's reported to date for elbow flexion and extension strength using anthropometric measures as predictors.

Based upon all analyses conducted, the present results strongly support the contention that, compared to segmental limb volumes, limb lengths and girths are better predictors of elbow flexion and extension strength in both male and female subjects. The difference in multiple R's obtained with and without segmental limb volumes are small and not significantly different. Anthropometric assessment techniques for estimating segmental limb volumes, moreover, require all of the anthropometric limb girth and length measures reported here to be obtained. As far as the results of this study are concerned, simple measures of limb girths and lengths, with and without body weight, are as good or better than segmental limb volume measures in the prediction of elbow strength in females and males. It is also rather clear

that the importance of anthropometric measures as strength predictors differed not only between the sexes, but between elbow flexion and extension strength as well.

LITERATURE CITED

AN, K.N., HUI, F.C., MORREY, B.F., LINSCHEID, R.L., AND CHAO, E.Y. 1981 Muscles across the elbow joint: a biomechanical analysis. J. Biomech. 14: 659-669.

BASMAJIAN, J.V. AND LATIF, S. 1957 Integrated actions and functions of the chief flexors of the elbow. J. Bone Joint Surg. 39A: 1106-1118.

BORCHARDT, J.W. 1968 A cluster analysis of static strength tests. Res. Q. 39: 258-264.

BUCHTHAL, F. AND SCHMALBRUCH, H. 1970 Contraction times and fibre types in intact human muscle. Acta Physiol. Scand. 79: 435-452.

CALDWELL, L.S., CHAFFIN, D.S. AND DUKES-DOBOS, F.N. 1974 A proposed standard procedure for static muscle strength testing. Am. Ind. Hyg. Assoc. J. 35: 201-206.

CLARKSON, P.M., KROLL, W. AND MELCHIONDA, A.M. 1981 Age, isometric strength, rate of tension development and fiber type composition. J. Gerontol. 36: 648-653.

CURETON, T.K. 1947 Physical Fitness: Appraisal and Guidance. St. Louis: Mosby.

DEMPSTER, W.T. 1955 Space requirements of the seated operator. WADC Technical Report 55-159. Wright-Patterson Air Force Base, Ohio.

ELBEL, E.R. 1949 Relationship between leg strength, leg endurance, and other body measurements. J. Appl. Physiol. 2: 197-207.

GOSLIN, B.R. AND CHARTERIS, J. 1982 The human elbow and knee: functional relationships and phylogenetic considerations. Hum Biol. 54: 491-500.

HETTINGER, T. 1961 Physiology of Strength. Springfield: C.C. Thomas.

HOFFMAN, T., STAUFFER, R.W. AND JACKSON, A.S. 1979 Sex difference in strength. Am. J. Sports Med. 7: 265-267.

IKAI, M. AND FUKUNAGA, T. 1968 Calculation of muscle strength per unit cross-sectional area of human muscle by means of ultra-sonic measurement. Int. Z. Ange. 26: 26-32.

JONES, H.E. 1947 The relationship of strength to physique. Am. J. Phys. Anthropol. 5: 29-39.

JOHNSON, M.A., POLGAR, J., WEIGHTMAN, D., AND APPLETON, D. 1973 Data on the distribution of fibre types in thirty-six human muscles: an autopsy study. J. Neurol. Sci. 18: 111-129.

KATCH, V. AND WELTMAN, A. 1975 Predictability of body segment volumes in living subjects. Hum. Biol. 47: 203-218.

KNAPIK, J.J. AND RAMOS, M.U. 1980 Isokinetic and isometric torque relationships in the human body. Arch. Phys. Med. Rehabil. 61: 64-67.

KROLL, W. 1972 Isometric bilateral reciprocal exercise. Arch. Phys. Med. Rehabil. 53: 515-522.

LAUBACH, L.L. AND MCCONVILLE, J.T. 1966 Muscle strength, flexibility, and body size of adult males. Res. Q. 37: 384-392.

LAUBACH, L.L. AND MCCONVILLE, J.T. 1969 The relationship of strength to body size and typography. Med. Sci. Sports 1: 189-194.

MORRIS, C.B. 1948 The measurement of the strength of muscle relative to the cross section. Res. Q. 49: 295-303.

MORROW, J.R. AND HOSLER, W.W. 1981 Strength comparisons in untrained and trained women athletes. Med. Sci. Sports Exerc. 13: 194-198.

NORDGREN, B. 1972 Anthropometric measures and muscle strength in young women. Scand. J. Rehabil. Med. 4: 165-169.

PETROFSKY, J.S. AND PHILLIPS, C.A. 1980 The effect of elbow angle on the isometric strength and endurance of the elbow flexors in men and women. J. Hum. Ergol. 9: 125-131.

PROVINS, K.A. AND SALTER, N. 1955 Maximum torque exerted about the elbow joint. J. Appl. Physiol. 7: 393-398.

RASCH, P.J. AND PIERSON, W.R. 1963 Some relationships of isometric strength, isotonic strength, and anthropometric measures. Ergonomics 6: 211-215.

ROBERTS, D.F., PROVINS, K.A., AND MORTON, R.J. 1959 Arm strength and body dimensions. Hum. Biol. 31: 334-343.

ROYCE, J. 1958 Use of body components as reference standards for basal metabolic rate. Res. Q. 29: 60-73.

RUPP, H. AND BARANOWSKI, P. 1934 Uber die kraft des menschen bei verschiedenen glieder-und korperstellung. Psychotechnise Z. 9: 40-52.

SADY, S., FREEDSON, P., KATCH, V.L. AND REYNOLDS, H.M. 1978 Anthropometric model of total body volume for males of different sizes. Hum. Biol. 50: 529-540.

SILLS, F.D., AND EVERETT, P.W. 1953 The relationship of extreme somatotypes to performance in motor and strength tests. Res. Q. 24: 223-228.

SINGH, M. AND KARPOVICH, P.V. 1968 Strength of forearm flexors and extensors in men and women. J. Appl. Physiol. 25: 177-180.

SMITH, L.E. AND ROYCE, J. 1963 Muscular strength in relation to body composition. Ann. N.Y. Acad. Sci. 110: 809-813.

TORNVALL, G. 1963 Assessment of physical capabilities. Acta Physiol. Scand. 58, Suppl 201.

WILKIE, D.R. 1950 The relation between force and velocity in human muscle. J. Physiol. 110: 249-280.

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